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HYPERPARATHYROIDISM

CLINICAL DIAGNOSIS AND THE OPERATIVE TECHNIQUE OF PARATHYROIDECTOMY

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THE clinical syndrome—hyperparathyroidism—due to excessive parathyroid secretion, as occurs with adenoma of the parathyroid gland, is now a well established entity. Rapid and pronounced relief of symptoms follows removal of the parathyroid adenoma. The differential diagnosis, however, is often confusing so that a true case of hyperparathyroidism may be missed or may even be unsuspected. The location and removal of the adenoma may present serious technical difficulties as evidenced by reports in the literature of cases operated upon several times in an attempt to find the adenoma.

In this paper five new proved cases of hyperparathyroidism are briefly reported, and the significant findings which led to the diagnosis are discussed. The operative exposure for removal of a parathyroid adenoma is illustrated, with particular emphasis upon the possible atypical location of parathyroid bodies—and so these tumors.

A consideration of the historical background of hyperparathyroidism is not included in this paper. (For references in this connection see bibliography.) It is, however, of interest to note a recent report by J. Bauer on further developments in the patient upon whom F. Mandl in 1926 performed the first operation to remove an adenoma of the parathyroid for relief of osteitis fibrosis cystica. Bauer reports, as in the original articles by Mandl, that the initial result was good and a formerly bedridden patient was able to walk without pain at the end of 2 months. This improvement lasted for 6 years. In 1932, however, this patient was admitted to Bauer's clinic because of a return of the original symptoms of pain on movement of the extremities, and because of progressive deformity of the skeleton. During hospitalization the patient developed renal colic caused by calculi. There was a hypercalcæmia (blood calcium 12.5 milligrams per hundred cubic centimeters), while the phosphorus was low (2.5 milligrams per hundred cubic centimeters). There was a negative calcium balance due to excretion of a large amount of calcium in the urine. A second operation was undertaken by Mandl because there appeared to be a return of the state of hyperparathyroidism. No tumor, however, was found. A subtotal thyroidectomy was therefore done. In this tissue Professor Sternberg found one normal parathyroid on the surface of, and another within, the body of the thyroid gland. There was no improvement following the operation. Bauer felt that symptoms persisted because of a hyperfunctioning adenoma of the parathyroid which, because of inaccessible location, was not found.

In view of the experience just related, as well as from a perusal of other cases reported in the literature, together with our own expe-

rience, it is obvious that pronounced technical difficulties are frequently encountered in attempts to locate and remove adenomata of the parathyroid gland.

Paget's disease of bones and parathyroid tumor. In view of this recent report on the further progress of Mandl's case, Kienbock states that a consideration of the entire course of this patient convinces him that the original diagnosis of Recklinghausen's osteitis fibrosa was erroneous. He believes this patient to be a case of Paget's disease of the bones associated with the unusual complication of a parathyroid tumor, with the bone changes as described, which in turn are rarely observed in Paget's osteitis. Emphasis is placed upon the absence of large cysts as seen in Recklinghausen's disease. Kienbock believes that the occurrence of the parathyroid adenoma in this instance was a coincidence rather than a cause of the condition. He further states that the mild form of hyperparathyroidism, as suggested by the studies of the blood chemistry in Mandl's case, is not infrequently seen in the porotic form of Paget's disease. Kienbock feels, however, that the two conditions, hyperparathyroidism and Paget's osteitis, are definitely distinct clinical entities. This conclusion is also stressed by W. Bauer. The authors, in 5 cases of Paget's disease, have at no time found reason to suspect the presence of a parathyroid adenoma because the clinical data are not similar to that found in hyperparathyroidism. X-rays do not reveal a generalized skeletal involvement. Clinically, Paget's disease of the bone is a different picture from that presented by hyperparathyroidism.

Chronic rheumatoid arthritis and hyperparathyroidism. It has been suggested that hyperparathyroidism is an etiological factor in the ankylosing type of chronic arthritis. One of us (GEH) reviewed the findings in 200 carefully studied cases of chronic rheumatoid arthritis. In no instance was the diagnosis of hyperparathyroidism confirmed. For example, the blood serum calcium and phosphorus estimations were consistently within normal range, while roentgenograms of the skeleton did not reveal the characteristic X-ray findings as described below.

DIAGNOSIS

The diagnosis of the clinical entity, hyperparathyroidism, depends primarily upon (1) an analysis of the chemical findings and (2) a careful survey of the roentgenograms, including not only those of the bones of the skeleton (especially plates of the skull, spine, pelvis, and femora) but also X-rays of the kidneys. The latter plates are taken to demonstrate the presence or absence of calculi. In very early cases of hyperparathyroidism, or in the presence of severe renal damage, study of the chemistry and X-ray findings may be misleading, and the diagnosis not arrived at.

From the clinical standpoint, cases of hyperparathyroidism may be conveniently classified in one of the following three groups:

1. Classical—the osteitis fibrosa cystica of von Recklinghausen's. Skeletal roentgenograms of these patients are quite characteristic, showing not only the diffuse granular osteoporosis, but also the presence of multiple cysts.

2. Hyperparathyroidism with osteoporosis—these cases are an early stage of group one and rarely show cyst formation. The decalcification of the bone is not as advanced as in group one, but there is present the characteristic osteoporosis, although at an early stage, as described below.

3. Cases in which renal pathology, due to the precipitation of calcium phosphate, is the significant finding. In these patients X-rays of the skeleton are often negative. Albright, Baird, Cope, and Bloomberg have subdivided this group of cases as follows:

a. Calculi in the renal pelvis, with resulting pyelonephritis. In a review of 38 cases there were 27 per cent in this group.

b. Calculi in the renal tubules, with resulting renal sclerosis, contraction, and insufficiency (23 per cent).

c. Precipitation of calcium phosphate in the kidney as one of several organs, with acute renal failure and death of undetermined cause in a few hours or days (1 per cent).

It is important to realize that renal lesions may occur without evidence of changes in the skeleton. The former are an index of the severity of the disease, and the latter an index to its duration.

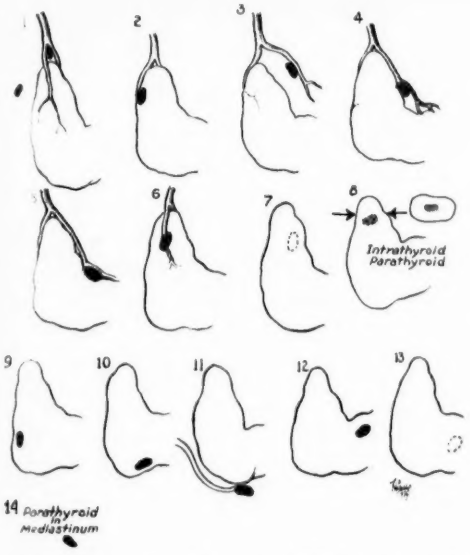

Fig. 1. On these diagrammatic illustrations of the anterior aspect of the thyroid, we have recorded the locations at which we have encountered parathyroids in the course of several thousand thyroid operations and the points where we have discovered bodies suspected and proved in 190 cases by microscopic section to be parathyroid bodies. These are the cases in which we have searched thyroid specimens while still sterile, in which we have found and transplanted microscopically proved parathyroids. Of all of these locations, that representing the superior parathyroid by dotted lines in *7* as being on the back of the upper pole where it rests against the larynx has been the most constant. The intrathyroid parathyroid as shown by barred lines in *8* is the location at which we have found parathyroid adenomata twice. Note in *7* a parathyroid shown in dotted lines on the posterior aspect of the thyroid resting against the trachea at the point where the isthmus and body join. Note in *14* the mediastinal location of a parathyroid and in *9*, *10*, and *12* parathyroids on the anterior aspect of the gland.

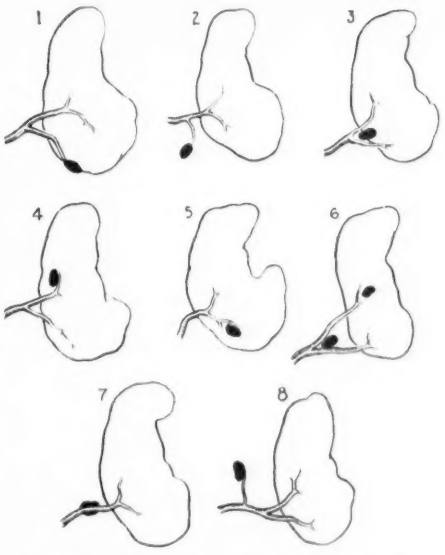

Fig. 2. These diagrammatic illustrations represent the thyroid gland pulled inward and exposing its entire posterior aspect as shown in the surgical illustrations Figures 9, 14, 19, 26, to 30 demonstrating the location of parathyroid adenomata at operation. On these are placed the locations at which inferior parathyroids have been demonstrated at thyroid operations and found and proved by microscopic section on the 190 parathyroid specimens removed and proved at the operating table. The most constant locations of parathyroid glands have been as shown in *3*, *4*, *5*, and *6*.

CLINICAL ASPECTS OF HYPERPARATHYROIDISM

The majority of the reported cases have occurred in females between 30 and 50 years of age. From an analysis of 115 proved cases (as detailed in the literature) Gutman, Swenson, and Parsons state that pain in the back or extremities was the major initial symptom in 72 per cent, while 22 per cent primarily complained of muscle weakness. The possibility of hyperparathyroidism must always be considered in patients who complain of relatively diffuse "neuritic" or "arthritic" pains, made worse by motion. This is particularly true if the symptoms exhibit localized or general bone tenderness, and if there is a history of pathological fracture. Not infrequently patients will report a progressive loss in stature (see Case 4).

The majority of the cases in the literature were reported to be in negative calcium balance. That is, these patients required a larger amount of calcium than the amount ordinarily believed to be adequate for maintenance of the normal individual in equilibrium. In early states of the disease, a negative balance may not be present, while in patients with bone involvement, from multiple myeloma or metastatic carcinoma, a definite negative calcium balance can often be demonstrated.

Classical hyperparathyroidism is characterized by an increased ratio of calcium in the urine to the total amount of calcium excreted,

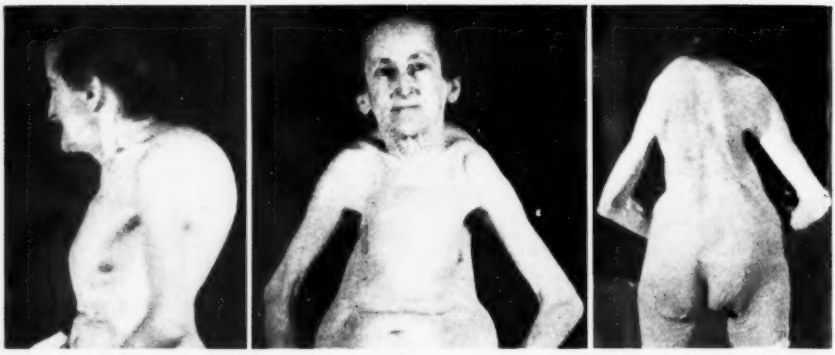

Fig. 3. Fig. 4. Fig. 5.

Figs. 3, 4, and 5. Case 1. Photographs of patient with hyperparathyroidism showing marked deformity of spine and thorax. Compare with Figures 7 and 8.

except in group 3 cases where the renal function is seriously impaired.

Blood chemistry. Classical hyperparathyroidism usually reveals an elevated blood calcium and relatively low phosphorus. The blood phosphatase activity is also increased. Taking the serum calcium value of 11.5 milligrams as a high normal, Gutman reviewed 78 cases in which the determination was recorded and found a blood calcium above 12 milligrams in 59 cases, while serum inorganic phosphorus determinations below 2.5 milligrams were found in 26 of 52 cases.

Fig. 6. Case 1. Roentgenogram of skull showing thickening of calvarium with loss of clear demarcation between the inner and outer table. The diffuse osteoporosis is well shown.

Obviously, hypercalcæmia and hypophosphatæmia are not consistently found in suspected cases. Particularly is this true in the presence of severe renal damage. Albright has shown that in these cases (group 3) the blood serum phosphorus rises, and the calcium tends to fall. Furthermore, Peters and VanSlyke have stressed the necessity of interpreting serum calcium values in view of the phosphorus and blood serum protein determinations.

In reviewing our knowledge of the blood phosphatase, Kay states it to be an enzyme present in the calcifying cartilage, and that its rise in hyperparathyroidism is a secondary effect. The source of the increased amount of blood phosphatase is not definitely known. Albright and his coworkers feel that this determination is an index of the degree of bone involvement, because in clinically proved cases of early parathyroid disease, without demonstrable bone change, the blood serum phosphatase is normal (normal being approximately three Bodansky units). Very high values have been found in active rickets (thirty to one hundred and sixty-five units) and in generalized Paget's osteitis (fifty to one hundred and thirty-five units). The same is

Fig. 7. Case 1. Anterior posterior roentgenogram of thorax illustrating deforming action of muscles of respiration on the softened bone.

Fig. 8. Case 1. Lateral roentgenogram of spine exhibiting pronounced crushing down of vertebral bodies with consequent loss in stature.

true in the blood serum of patients with obstructive jaundice.

X-ray findings. If the diagnosis of hyperparathyroidism is suspected, X-ray studies should include plates of the skull, spine, and pelvis. In addition to roentgenograms made of the extremities presenting symptoms, plates of the femora and humeri are also helpful in detecting early bone change. The characteristic changes in the skeleton, together with comparable pathological findings as described by Hoffheinz are outlined as follows:

Skull (Figs. 24, 27). There is a definite thickening of the calvarium, together with a pronounced blurring of outline, so that the demarcation between inner and outer tables is very poorly defined, at times lost. The bones present a finely mottled granular appearance, that is, a diffuse osteoporosis. In the more advanced cases there are large irregular areas of diminished density, and these are often more pronounced in the mandible and maxilla.

From observations at necropsy Hoffheinz found the surface of the bones of the skull to be very rough, with numerous irregularly spaced sharp pointed projections, between which were depressions averaging 2 to 3 millimeters in size. Also seen on the bone surface were scattered depressed areas 2 to 3 centimeters in diameter with adjacent osteophytic deposits. Most of the bone was sufficiently soft to be easily cut with a knife. The inner and outer tables could not be recognized on section.

In a chemical analysis of bone from a patient with hyperparathyroidism, Wilder found considerable loss of calcium and phosphorus and a relative if not an absolute gain in organic matter.

Vertebræ. The vertebral body of advanced cases shows a pronounced crushing or narrowing, particularly at the anterior margin (Fig. 8). In less severely affected vertebræ there is an increase in the convexity of the superior and inferior body surfaces. Such deformity, affecting many vertebral segments, results in a pronounced kyphosis, thus causing a marked increase in the normal anterior posterior curve of the spine, as well as a loss in stature (Fig. 8). The vertebral body in some instances presents a finely mottled appearance, with coarse perpendicular striæ. In other cases (Fig. 29), there is a diffuse, rather even,

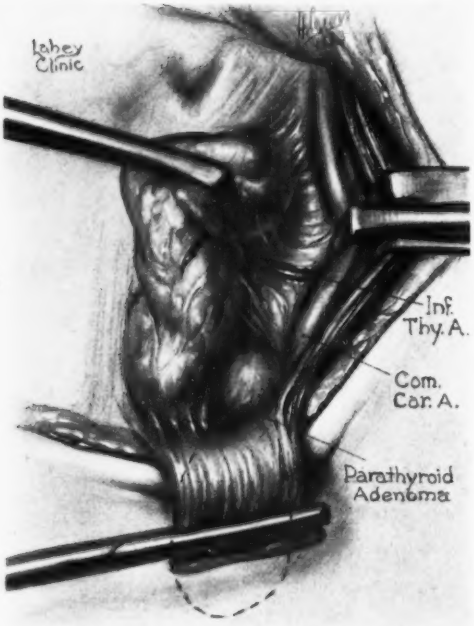

Fig. 9. Case 1. In this drawing, made by the artist at the operation, the top of the large intrathoracic parathyroid adenoma may be seen appearing just above the level of the clavicle and the size of the intrathoracic portion outlined by dashes upon the chest wall. The shadow of this intrathoracic parathyroid tumor is demonstrated in the accompanying X-ray film of this patient's superior mediastinum. Note the high position of the inferior thyroid artery. We have called attention to the not infrequent high course of this vessel. The recurrent laryngeal nerve was safely located as it so often is in intrathoracic thyroid adenomata posterior to the intrathoracic parathyroid adenoma so that it was not injured.

decalcification of the entire bone. The intervertebral discs are not affected.

In connection with the spine, Figure 7 well illustrates the deforming action of the muscles of respiration on the softened thoracic cage, a deformity analogous to that seen in severe rickets.

Pelvis. The pelvic bones exhibit changes similar to those seen in the skull. In many instances, the pelvis is markedly deformed, presenting a wedge-shaped appearance with large cystic areas scattered throughout the bone.

Bones of the extremities. Figure 12 shows the femora of Case 2, upon which X-ray films the diagnosis of hyperparathyroidism was made, to be later confirmed by chemical analyses and operative findings. These plates are typical of the early bone change (type 2) and illustrate a slight but definite diffuse osteoporosis of the femora.

Compare the plates mentioned with those of Figure 18 which illustrates bone change in the femur of an advanced case of hyperparathyroidism of long duration. Here is seen evidence of old healed spontaneous fractures. The cortex of the bone is exceedingly thin. There is a pronounced generalized demineralization, so marked, indeed, that the mottled appearance is not evident. Where cysts are found, expansion of the cortex occurs. There is no periosteal proliferation.

The characteristic subperiosteal rarefaction of the cortex and the formation of small cysts are usually best shown at the metaphysis of the long bones and on the superior and inferior margins of the ribs.

The pathological studies of Hoffheinz reveal that the bones of the extremities show much less evidence of alteration of structure than is the case in the bones of the skull. In the femora and humeri, even in advanced cases, there is a clear differentiation between the cortex and the spongy bone.

Location of bone changes. The site of the bone lesions in hyperparathyroidism may be explained by the work of Jaffe, Bodansky, and Blair through their investigations on guinea pigs, dogs, and rats. These animals were given large doses of parathormone with the resulting specific effect of decalcification of the bones. The resorption of bone was established by histological study, which revealed the presence of large haversian canals and of lacunæ containing osteoclasts on the periosteal and endosteal surfaces of bone.

The authors point out that bone resorption and bone deposition is a process that constantly goes on in all bone, and that these processes are more rapid in the regions of active bone growth. If the rate of either resorption or deposition of bone is increased, it is increased throughout the animal body. Hence, if a condition is present favoring an excess of resorption of bone, as hyperparathyroidism, such resorption will be most evident in the regions of active bone growth. A pronounced

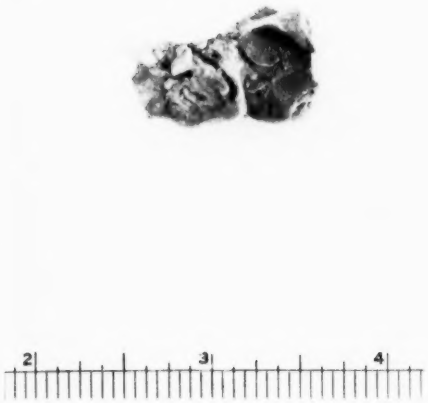

Fig. 10. Case 1. Photograph of tumor removed.

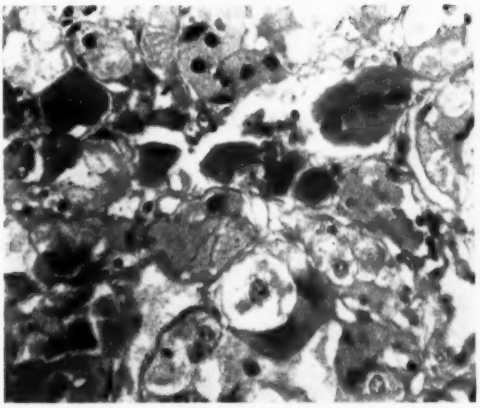

Fig. 11. Case 1. Photomicrograph of parathyroid adenoma. ×475.

stimulus, as hyperparathyroidism of long duration, would be necessary to affect the whole skeleton.

The above theory would then explain the pronounced susceptibility to resorption of the spongy bone of the metaphyses, and of the cortical shaft in the epiphyseal regions in the rapidly growing long tubular bones; as well as resorption at the costochondral junctions of the ribs, the rib cortex, and in the bones of the skull.

Summary of X-ray findings. The bone changes in hyperparathyroidism as shown by roentgenograms may therefore be summarized as follows:

1. In the relatively early cases there is a diffuse osteoporosis. The bone exhibits a finely mottled granular appearance, or there may be a pronounced general demineralization of the bone. In the more advanced cases there are large irregular areas of diminished density with cyst formation. The cortex of the long tubular bones is exceedingly thin, particularly in the epiphyseal regions. No periosteal reaction is seen.

2. In long standing cases bone deformity is present, viz.: a pronounced kyphosis of the spine, due to crushing down at the anterior margin of many vertebral bodies; deformity of the ribs, the result of action of muscles of respiration on the softened thoracic cage; wedge-shaped pelvis; and finally, the deformities following spontaneous fracture of the long bones.

With the diagnosis of hyperparathyroidism established, the problem then relates itself, for the most part, to the question of finding the parathyroid adenoma and removing it. Albright, Bauer, and Churchill have recently reported cases of hyperparathyroidism apparently not due to adenomata of the parathyroids but rather to hyperplasia of all the parathyroids. This finding, as we have previously stated, complicates the management of hyperparathyroidism and, if proved by further experience with this condition to be convincingly true, may explain some of the cases in which search for a parathyroid tumor has proved unsuccessful.

Failure to find parathyroid adenomata in many cases will be due to failure to realize the frequent atypical location of the parathyroids and failure to investigate the possible atypical locations for a possible parathyroid adenoma.

It becomes also the duty of pathologists, in view of this report from a group of men who have done such sound work in connection with this disease, to familiarize themselves with the normal and abnormal pathology of the parathyroid gland, since it will be necessary in cases in which adenomata cannot be found to call the pathologists into the operating room for opinions on frozen sections: first, as to whether removed specimens are, or

Fig. 12. Case 2. Note the slight but definite diffuse osteoporosis of these femora. (Type 2.)

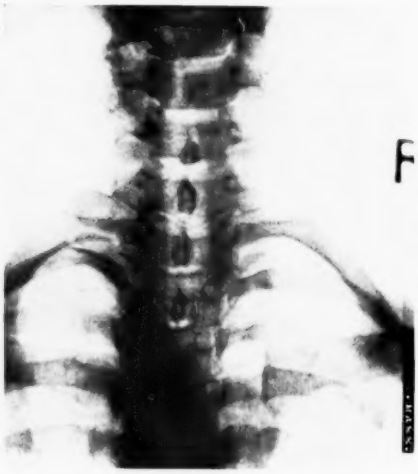

Fig. 13. Case 2. Anterior posterior X-ray pictures of cervical spine which show early even generalized decalcification of the bones to be compared with Figure 12. In this plate definite hypertrophic arthritic change is also seen. The one patient in this series in which these findings were observed.

are not, parathyroids, and second, whether or not parathyroid hyperplasia, if adenomata are not found, comparable with that in hyperthyroidism exists, and whether or not on such evidence, combined with the clinical findings, subtotal parathyroidectomy is justified. With these ideas in mind, our pathologists have undertaken a review of the histology of all of our fixed and preserved parathyroid specimens, amounting to one hundred and ninety specimens, each representing a section of an individual parathyroid—their report to be published later.

As the result of the plan which we have published previously,[1] of searching every thyroid specimen surgically removed for possible parathyroids and of reimplanting the removed parathyroid body into the left sternomastoid muscle, a large number of parathyroid glands have been found on the removed thyroid gland. Due also to the policy before transplantation of cutting off a small gross section of the suspected parathyroid gland and submitting it to the pathologist for report, we have acquired a relatively large mass of parathyroid material and a microscopically proved experience in deciding as to what is and what is not a parathyroid, with variation in shape, size, color, and location of these glands.

The parathyroid most easy to distinguish has a brownish color, varying from dark to light mahogany brown. It has molded edges, quite similar to the molded edges seen in a miniature lima bean. In fact, one of us (FHL) has often stated that, except for the fact that they are small, and tend to be convex on one side and concave on the side in contact with the thyroid, the typical non-adenomatous parathyroid more closely resembles a miniature lima bean (except as to color) than anything else.

As the result of our experience now with over 13,000 thyroid operations, in all of which the regions where parathyroids are found are widely exposed and when possible parathyroids demonstrated, we have learned that there is considerable variation in size in the non-adenomatous parathyroid glands from that of the head of a match up to the size of a small lima bean. We have likewise learned

[1] Frank H. Lahey. The transplantation of parathyroids in partial thyroidectomy. Surg., Gynec. & Obst., 1926, 42: 508-509.

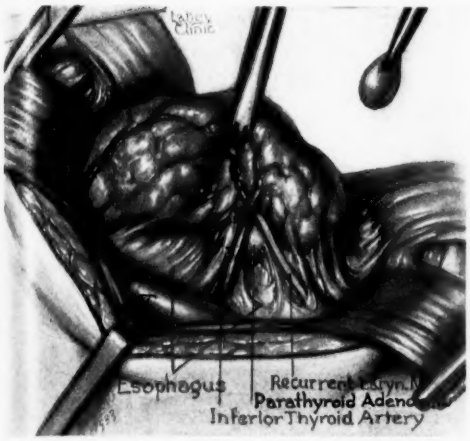

Fig. 14. Case 2. The parathyroid adenoma as shown in this illustration made at the time of operation was found in the groove between the trachea and œsophagus behind the trunk of the inferior thyroid artery and beneath the recurrent laryngeal nerve. This is not an uncommon location for parathyroids. As may be seen in the diagrammatic illustrations, Figures 1 and 2, showing the points on the back of the thyroid gland where parathyroids have been found in thyroid operations, should an adenoma occur in the parathyroid shown in Figures 3, 6, or 7 it would be located in just the same position as was the one in this case. The tumor shown in the jaws of the hæmostat is the actual size of the parathyroid adenoma on removal.

The course and relationship of the inferior thyroid artery and recurrent laryngeal nerve as shown here is the one which we most commonly see in our thyroid operations.

Note in all of these cases that the prethyroid muscles have been cut, the gland pulled inward with thyroid grasping forceps, and its posterior aspect widely exposed.

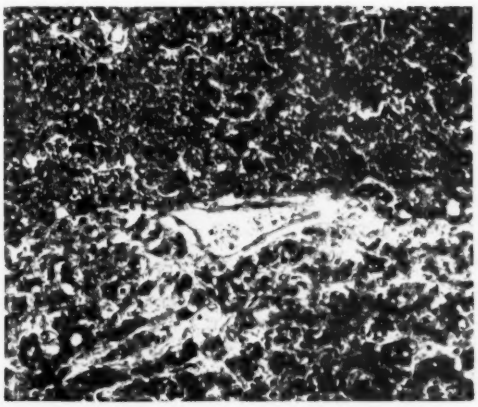

Fig. 15. Case 2. Photomicrograph of parathyroid tumor tissue. ×140.

that the back of the thyroid gland (in contact with which gland the parathyroid glands commonly rest) is sometimes quite free from yellow fatty tissue, so that brown parathyroids are readily located and distinguished, while at other times this region is thickly sprinkled with fat, particularly about the point where the branches of the inferior thyroid artery penetrate the gland, and where inferior parathyroid glands are so often demonstrable, thus making the demonstration of these bodies less certain.

Such parathyroids are sometimes overlaid by adipose tissue and are yellow or pale white in color. In such fat infiltrated parathyroids one of us (FHL) has noted the tendency of these glands to be more globular in shape and to have lost the molded edges characterizing the more typical, more easily distinguished glands.

Early in our experience with parathyroid bodies, we had impressed upon us, and remarked it in our publications on this subject, that these glands were often atypically located and that they might be found anywhere—on, in, behind, or below the thyroid gland. During one of his visits with us Dr. Wallace I. Terry became interested in our demonstration of atypically placed parathyroids and delegated one of his junior men, Dr. Raymond J. Millzner, to search all specimens for such atypically located parathyroid glands, with the result that valuable reports concerning such atypical locations have also emanated from Dr. Terry's clinic in San Francisco. Recently, with the interest intensified in hyperparathyroidism, reports are frequently appearing in this country and abroad concerning atypically located parathyroid bodies which have become the site of parathyroid adenomata.

The two most constant points at which we have found parathyroid bodies have been on the back of the gland, close to the point at which the inferior thyroid artery enters the gland and below the lateral exit of the middle and lower thyroid veins (the inferior parathyroids); and on the posterior lateral aspect of the upper pole of the thyroid where it rests against the laryngeal cartilage (superior parathyroid).

We have seen microscopically proved parathyroid bodies atypically situated so often in

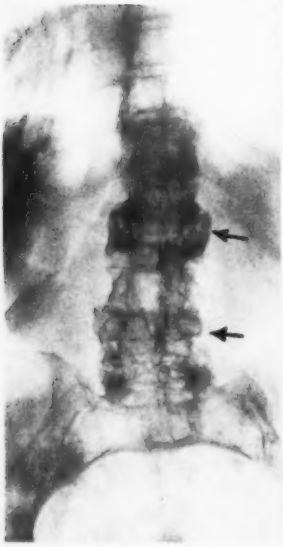

Fig. 16.

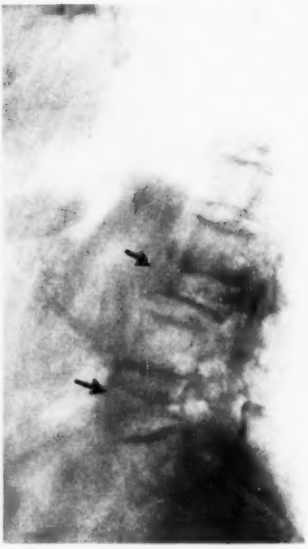

Fig. 17.

Fig. 18.

Fig. 16. Case 3. Roentgenogram of lower spine and pelvis. A long standing case of hyperparathyroidism with consequent bone deformity. Compare with Figures 17, 18.

Fig. 17. Case 3. Lateral X-ray film of lower spine illustrating marked crushing down of vertebral bodies with old healed fracture.

Fig. 18. Case 3. Compare with Figure 12. Here are shown the bone changes in the femur of an advanced case of hyperparathyroidism of long duration. The old but now healed spontaneous fractures are evident. The cortex of the bone is exceedingly thin. Generalized demineralization is seen. There is no periosteal proliferation.

a variety of locations that it has seemed best to illustrate their possible location by placing them on diagrammatic outlines of the thyroid seen from an anterior, Figure 1, and posterior, Figure 2, view. We have seen parathyroids in the mediastinum (parathyroid adenomata and on intrathoracic goiters), on the side of the thyroid gland, on the front of the thyroid gland, in the bundle of vessels entering the upper pole, in the groove between the trachea and œsophagus, well away from the gland, out on the trunk of the inferior thyroid artery before it had divided, and within the substance of the thyroid gland itself. Dr. R. B. Cattell, working in the Clinic in 1924 on the relation of the histology of the thyroid to the administration of iodine (at which time sections were made of 400 hyperplastic thyroid glands, removed at operation), first called our attention to the relative frequency with which intrathyroid parathyroids were found within the specimen removed by subtotal thyroidectomy.

As the result of a large experience (190) with the microscopically proved parathyroids at thyroid operations and on the specimen immediately after the operation with the specimen still sterile, we believe we can safely state that a parathyroid may be found at practically any position on, in, behind, or below the thyroid gland.

The technique of demonstrating and removing parathyroid adenomata is related largely to exposure of the region where parathyroid bodies are found when typically located and a knowledge of the regions in which to search for them when parathyroid adenomata are not demonstrated in typical locations.

The first requirement of any operation for removal of a parathyroid adenoma is exposure. This involves in our hands an adequate thyroid incision with a severing of the prethyroid muscles high between clamps. Without these muscles severed, we are not able to obtain wide exposure of the back of the gland from its lowest point up to its upper pole

Fig. 19. Case 3. In this illustration made at the time of operation one sees the upper pole of the thyroid gland split where it rests against the larynx, and an intrathyroid parathyroid demonstrated within the substance of the thyroid gland. By pressing the upper pole of the thyroid gland against the thyroid cartilage, this small tumor could be palpated as a bean-like structure within the gland, demonstrated, removed, and immediately proved by frozen section to be a parathyroid adenoma.

As stated in the text, in a case seen in consultation, see Figure 32, a and b, in which two previous unsuccessful searches for a parathyroid adenoma had been made, at our suggestion a careful search at the third operation for an intrathyroid parathyroid revealed one and permitted its removal. Where definite chemical, clinical, and X-ray evidence of hyperparathyroidism are present but the parathyroid tumor not evident on operative exposure, careful palpation of the thyroid gland must be made with the possibility that the parathyroid adenoma is within the gland.

In the diagrammatic drawing (Fig. 1) showing the locations of parathyroids from the front of the gland, this position is shown in *8*. This should not be a very uncommon location for parathyroid adenomata since *7* shows in dotted lines the location in our experience of the most constantly placed superior parathyroid just behind the internal and lateral aspect of the upper pole where it rests against the thyroid cartilage.

With the muscles severed the middle thyroid veins are ligated between clamps so that the lateral vascular attachments of the thyroid to the internal jugular vein are severed. The outer border of the thyroid gland is then grasped with the thyroid grasping forceps,

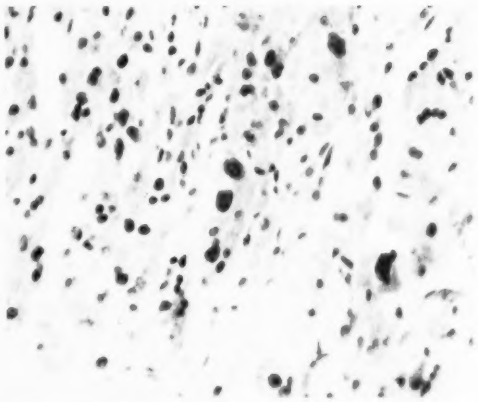

Fig. 20. Case 3. Photomicrograph of parathyroid adenoma from this patient. ×500.

which we have described, lifted up, and rotated inward. The internal jugular vein and common carotid artery are completely separated from the back of the thyroid gland and the inferior thyroid artery completely demonstrated as a trunk where it passes behind the common carotid artery. The back of the gland is inspected for a tumor body on the gland, and, if a tumor cannot be seen, it is palpated for such a tumor within the gland. An adenoma of a parathyroid usually converts that structure into a globular mass and changes its color to a pale white rather than the brownish color characterizing the typical normal parathyroid.

Parathyroids, involved or uninvolved by adenomata, are characterized by being so movable on the back of the thyroid that they are demonstrably not part of that gland. On removal they have a glistening covering, entirely surrounding the removed structure, with no area over which this shining covering is absent. This is of value in distinguishing a parathyroid adenoma from an extruded adenomatous thyroid area, in that the latter structure is usually connected with the thyroid itself by an isthmus of thyroid tissue, the severing of which in its detachment from the thyroid leaves a cut surface of thyroid tissue—interrupting thus the smooth and complete glistening capsule which is to be found in both parathyroids and parathyroid adenomata.

Fig. 21.

Fig. 21. Case 4. Photograph of patient illustrates the upper dorsal round back with consequent loss of stature due to crushing down of vertebral bodies as illustrated in roentgenograms of the spine, see Figures 22 and 23.

Fig. 22.

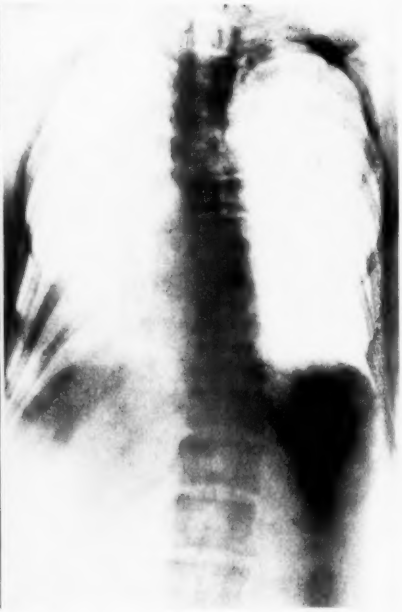

Fig. 23.

Figs. 22 and 23. Case 4. Anteroposterior and lateral roentgenograms of spine. The body of the fifth dorsal vertebra is represented only by a thin wedge of bone. The patient stated there has been progressive loss in height.

Should no parathyroid adenoma be demonstrable on this exposure, the inferior thyroid artery must be completely dissected up to its entrance into the thyroid gland. The recurrent laryngeal nerve must be found and dissected up to its passage behind or in front of the inferior thyroid artery. We have found and removed a parathyroid adenoma at this point behind the recurrent laryngeal nerve and inferior thyroid artery (Fig. 14).

If still no parathyroid adenoma is found, one must expose the groove between the trachea and œsophagus in the region of the inferior thyroid artery. If still the adenoma is undemonstrated, the superior thyroid artery should be ligated, and the upper pole of the thyroid turned downward, so that the region where it rests against the thyroid cartilage may be inspected. In dissections at this point troublesome bleeding may arise from the ascending branch of the inferior thyroid artery, a branch of which usually nourishes the upper parathyroid. In controlling bleeding at this point, caution must be exercised, as the recurrent laryngeal nerve enters the larynx here, at the point where the lowest fibers of the inferior construction are inserted into the horn of the thyroid cartilage shown at × in Figure 26.

In the event that the tumor is still not found, one palpates the thyroid through and through between the thumb and the forefinger and any discrete globular nodule within the gland is exposed by an incision into the gland, removed, and submitted to the pathologist for immediate frozen section report as to a possible intrathyroid parathyroid. We have removed such an intrathyroid parathyroid as shown in Figure 19.

The X-ray picture of the bones of another patient is shown in Figure 32, a and b, demonstrating to what an extreme state decalcification can extend in hyperparathyroidism. This patient was seen in consultation with Dr.

Fig. 24. Case 4. The generalized decalcification and soft mottled appearance of the skull is characteristic.

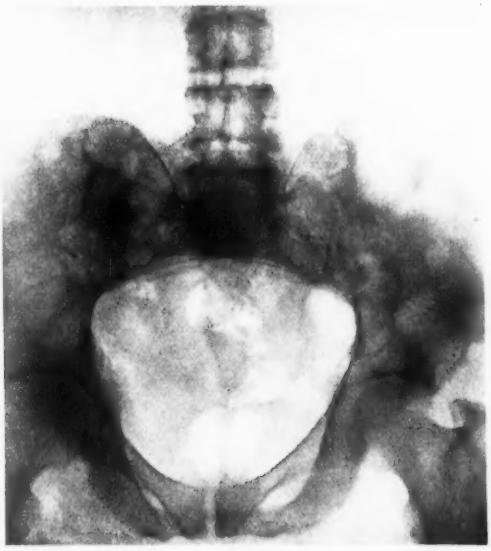

Fig. 25. Case 4. Roentgenogram of pelvis illustrating the multiple large cystic areas seen in these bones.

Robert C. Cochrane at the Boston City Hospital. She was a case of proved hyperparathyroidism and had previously had two normal parathyroids removed without relief of symptoms. We advised another operation and a search within the thyroid gland itself. Such a search revealed an intrathyroid parathyroid adenoma which was removed.

With failure still to demonstrate the parathyroid tumor, the search is continued in the bundles of the superior thyroid vessels, out along the main trunk of the inferior thyroid artery, behind the œsophagus and along the inner aspect of the upper pole. The isthmus is palpated against the trachea (a parathyroid has been reported as having been found on the posterior aspect of the isthmus). If a pyramidal lobe is present it is exposed and palpated throughout its extent.

If the evidence of the existence of hyperthyroidism be certain by clinical and laboratory evidence, and still no parathyroid tumor has been found, and if digital palpation of the superior mediastinum demonstrates no palpable parathyroid tumor, the manubrium of the sternum may be removed to facilitate a visual search for a mediastinal parathyroid adenoma since we have, and Churchill likewise has, demonstrated and removed a parathyroid adenoma from the superior mediastinum.

Should exposure of all these regions fail to demonstrate an adenoma of a parathyroid, it then becomes justifiable to remove and submit a normal appearing parathyroid to the pathologist for decision as to the possible pressure of hyperplasia of the parathyroids.

A brief abstract of the cases of hyperparathyroidism is submitted below, together with X-rays of the bones, illustrations of the location of the parathyroids drawn by the artist at the time of removal, the actual size of the adenoma of the parathyroid, a microscopic section of the tumor, and follow-up notes as to their postoperative course and changes in blood chemistry.

CASE REPORTS

Case 1. Mrs. Rebecca B., aged 62 years, housewife. April 17, 1933. Patient came to the Clinic because of a progressive increasing deformity of the upper spine, a round back, and a loss of stature of some 5 years' duration. The degree of this deformity is well illustrated in Figures 3, 4, and 5. There were likewise multiple diffuse generalized aches and pains.

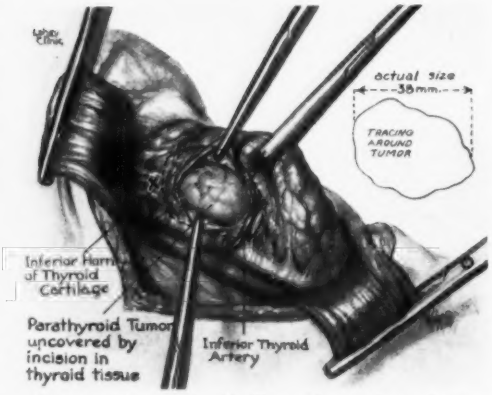

Fig. 26. Case 4. In this illustration made at the time of operation, a tracing of the outline of the removed parathyroid adenoma is shown demonstrating thus the actual size of the tumor.

Note the location of this tumor just below the horn of the thyroid cartilage suggesting that this adenoma probably arose in the superior parathyroid where it so commonly rests against the thyroid cartilage.

This tumor was entirely covered by thyroid tissue but could be moved upward and downward within the gland. Its relation to the recurrent laryngeal nerve which enters the larynx indicated by *x* at the level of the horn of the thyroid cartilage is to directly overlie it.

X-ray examination revealed the classical picture of a relatively advanced hyperparathyroidism. Figures 6, 7, and 8 are films of skull and spine showing the typical changes as described.

The initial blood calcium was 17.2 milligrams, the blood phosphorus 2.5 milligrams, and the phosphatase activity 64.4. Pre-operative estimation of these values showed an average calcium of 13.3 milligrams, and a phosphorus varying at times from 2.3 to 1.7 milligrams. The blood non-protein nitrogen averaged 30 to 35 milligrams. The phenolsulphonphthalein test showed a kidney function of 25 to 35 per cent. There was a very slight trace to a slight trace of albumin in the urine, with some slight fixation of the specific gravity by 2 hour examination. It was evident that there was some renal damage. The basal metabolism was plus 20. On a fixed amount of calcium intake the patient was shown to be in negative calcium balance.

Operation was done on May 15, 1933. A parathyroid adenoma was excised (FHL). Beneath the lower left pole of the thyroid there was a definite tumor mass which descended into the chest. This mass was 1.5 inches long and about three-quarters of an inch in diameter (Figs. 9 and 10). It was distinctly separate from the thyroid gland, except for some connecting alveolar tissue and blood vessels. An immediate frozen section revealed parathyroid tissue, and further histological study confirmed the diagnosis of an adenoma of the left lower parathyroid gland (Fig. 11).

Following operation, the patient developed definite tetany on the evening of the third postoperative day. Blood calcium at this time was 8.5 and the blood phosphorus 1.2. Intermittent signs of tetany, severe at times, persisted until the thirteenth postoperative day. The tetany was controlled by administration of parathormone and large doses of calcium lactate by mouth. Viosterol capsules were also given. On discharge from the hospital she was instructed to take 5 grams of tribasic calcium phosphate daily and continue with the viosterol, drops x, twice daily at this time. The blood calcium was then 7.8 milligrams and the phosphorus 2.4 milligrams.

November 17, 1933. Six months following operation blood calcium was 9.5 and the phosphorus 3 milligrams. The phosphatase activity was 7.5. The patient was entirely free of her aching discomforts in the back and extremities. She had taken calcium phosphate in small doses up until this time and showed no evidences of tetany.

December 3, 1934. Eighteen and one-half months following operation the blood serum calcium was 9 milligrams and phosphorus 4 milligrams. The phosphatase activity was 3.7 Bodansky units. The patient had not taken calcium for 1 year. She revealed no signs of tetany and was entirely free from any symptoms.

Case 2. Mrs. Anita K., aged 53 years, housewife. This patient is of particular interest in that she is the only one of the group here reported, presenting definite evidence of hypertrophic arthritis associated with hyperparathyroidism.

May 2, 1933. She was admitted to the clinic because of painful knees and pain in the back of the neck region of 4 to 5 years' duration, slowly but progressively becoming more pronounced. She had been treated elsewhere without relief by tonsillectomy, medication, and various other forms of diet.

The significant clinical findings were as follows: A well developed, fairly well nourished, very tired and worn appearing Armenian woman of 53 years with moderate upper dorsal round back, crepitation in knee joints, plus some periarticular thickening, and definite limitation of motion of the head due to pain. These symptoms were at first thought to be due simply to pronounced fatigue, associated with a moderately advanced hypertrophic arthritis. X-rays of the knees, showing the lower femora, however, revealed a diffuse, although not pronounced osteoporosis (Fig. 12). Parathyroid disease was therefore suspected. X-rays of the cervical vertebræ (Fig. 13) were even more suggestive of the same condition, although they also revealed extensive hypertrophic changes. Plates of the skull were negative. No tumor could be felt in the neck. The patient was then advised to enter the hospital for investigation of possible hyperparathyroidism.

The blood calcium ranged from 12.5 to 14.9 milligrams; the phosphorus 3 milligrams to 2.2 milligrams. Careful metabolic studies revealed a definite negative calcium balance. The urine calcium ex-re-

Fig. 27. Case 4. Photograph of parathyroid adenoma.

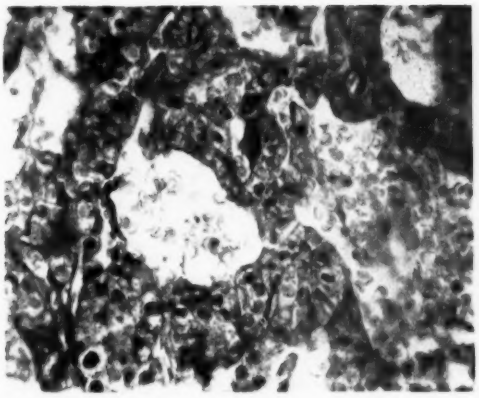

Fig. 28. Case 4. Photomicrograph of parathyroid adenoma. ×500.

tion was 0.328 grams to 0.423 grams. The blood phosphatase activity, however, averaging 10.2 units was approximately normal, but in view of the relatively insignificant bone changes, this phosphatase finding was not felt to contra-indicate definitely the diagnosis of hyperparathyroidism. This patient is classified in group 2 and in view of the hypercalcæmia and X-ray findings described, exploration of the neck was advised.

June 7, 1933, operation was done (FHL). A definite mass about equal to a medium sized olive was felt beneath the right inferior thyroid artery. Following demonstration of the recurrent laryngeal nerve, the inferior thyroid artery was ligated and the tumor mass revealed. With very careful dissection, it was freed from the nerve, delivered, and removed (Fig. 14). An immediate frozen section diagnosis of parathyroid adenoma was made and later confirmed by further microscopic study (Fig. 15).

On June 24, 1933, 17 days following operation, the blood calcium had come down from an initial high reading of 14.9 to 9.7 milligrams, while the phosphorus had risen from an initial low reading of 2.2 milligrams to 3.8 milligrams. The urine calcium with an original high reading of 0.462 grams diminished to 0.109 grams.

At no time was there evidence of postoperative tetany. The patient's aches and pains, however, persisted, especially in the neck and knee region until September 1, 1933, 3 months following operation, when symptoms were much less pronounced.

September 19, 1934, 15 months following operation blood serum calcium 10.7 milligrams, phosphorus 3.5 milligrams. Patient reported that aching discomfort was still intermittently present in the neck region, but the severity of these pains was much less pronounced. The knee joint symptoms had disappeared. She felt greatly improved over preoperative condition.

Case 3. Mrs. Elloise S., aged 74 years, housewife. The diagnosis of hyperparathyroidism in this patient rests upon the history; the X-ray findings, and upon the operative and microscopic demonstration of an adenoma of the parathyroid gland. The blood chemistry was normal. Metabolic study showed a slight negative calcium balance but no more than might be present in conditions other than hyperparathyroidism.

June 2, 1933, she was admitted to the clinic with a complaint of goiter, nervousness, and severe leg pains. Patient gave a history indicative of three spontaneous fractures in the region of the right knee and the right ankle and left ankle during the past 6 years. All had healed, but considerable deformity resulted, particularly in the region of the right knee.

Figures 16 and 17 are X-ray films of the spine and show marked decalcification with pronounced compression of the eleventh dorsal and second and fourth lumbar vertebræ. Small cystic areas are seen in the pelvis. Plates of the femora (Fig. 18) show an old malunited fracture of the distal end of the right femur, as well as a more recent fracture of the medial condyle of this femur.

The highest blood serum calcium finding was 11.3; the average of 4 estimations was 10 milligrams per hundred cubic centimeters. The blood phosphorus varied from an initial reading of 1.9 to 3 milligrams. The blood phosphatase activity was 7.2.

June 8, 1933, operation was done (FHL). The left thyroid lobe was occupied by a discrete adenoma, the size of a lime, and a small adenoma about the size of a cherry, both of which were removed, and on examination presented the typical gross and microscopic appearance of multiple colloid adenomatous goiter. Very careful palpation of the atrophic right lobe of the thyroid revealed a small discrete tumor about a centimeter in diameter in the substance of the gland at its superior pole. The actual size of this adenoma of the parathyroid may be seen in Figure 19, made by the artist at the operating table. This mass was therefore removed and on immediate frozen section showed parathyroid tissue. Further microscopic study revealed an adenoma of the right upper parathyroid gland (Fig. 20).

The postoperative course was quite uneventful. There was at no time any suggestion of tetany.

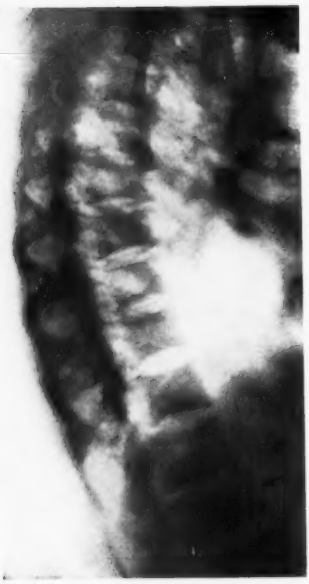

Fig. 29. Case 5. Lateral X-ray roentgenogram of spine showing the generally diffuse even decalcification of the column. Crushing of the body of first lumbar vertebra suggestive of fracture but there was no history of trauma. Note absence of involvement of intervertebral discs.

August 11, 1933, patient reported for check up examination at which time the calcium was 10.2 and the phosphorus 3.3 milligrams. The aches and pains in the legs were considerably improved. She was still having symptoms referable to the mechanical defect resulting from the malunited fracture, for which the patient refused any further therapy. Further studies of the renal function at this time as well as investigations before operation did not reveal definite evidence of a renal insufficiency. The urine remained negative and the blood non-protein nitrogen was 30 milligrams.

January 24, 1934, 6 months postoperative, the patient reported that she was not bothered by the leg pains except those due to old fracture of the right knee region. December 7, 1934, 18 months following operation the blood serum calcium was 10.0 and the phosphorus 4.1 milligrams. The blood phosphatase was 3.3 Bodansky units. The original complaint of nervousness and leg pains no longer bothered patient.

Case 4. Miss Rachel E., aged 44 years, clerk. This patient was originally admitted to the clinic in January, 1930, for a multiple colloid adenomatous goiter, partly substernal, without evidence of toxicity. Excision of the goiter was advised and carried out in October, 1930, and the patient made an uneventful recovery. At this time there was no evidence of an adenoma of the parathyroid gland, nor were there any clinical signs of hyperparathyroidism.

The patient next returned to the clinic in June, 1934, because of diffuse low back pain and pain in the knees and legs of 2 years' duration. In this interval, particularly in the past 10 months, the patient described a progressive loss of stature with an increasing round back deformity. Figure 21 is a photograph of the patient at this time. There was no evidence of a tumor in the thyroid region.

Figures 22 and 23 are lateral and anteroposterior X-ray films of the spine which reveal practically complete destruction of the fifth thoracic vertebra from marked compression. The pronounced deformity of the thorax is evident. A lateral view of the skull (Fig. 24) shows typical gross decalcification, with prominence of the trabeculæ and the soft mottled appearance of hyperparathyroidism. Numerous multilocular cysts as found in advanced cases of classical form of the disease are illustrated in the X-rays of the pelvis (Fig. 25). X-rays of the genito-urinary tract revealed the kidneys to be of normal size and regular in outline. There was no evidence of stone.

Initial blood serum calcium values ranged from 14.4 to 15.1 while the serum phosphorus varied from 2.4 to 3.5 milligrams. The phosphatase activity was 35 Bodansky units. The remaining findings from the complete blood study were negative. The basal metabolism was +14.

Operation was done July 9, 1934 (FHL). A tumor mass was found in the lower lateral side of the remnants of the right lobe of the thyroid gland. This tumor, about the size of a large walnut, lay partly beneath the inferior thyroid artery. In Figure 26 is shown an actual outline tracing around the parathyroid adenoma. Immediate frozen section diagnosis was adenoma of the parathyroid which conclusion was later confirmed by paraffin sections. Figure 27 is a part of the parathyroid adenoma removed and Figure 28 is a photomicrograph of the adenoma.

The postoperative course was uneventful. The blood calcium immediately came down to 9.1 and the phosphorus to 3.1 milligrams. Two weeks after operation, the serum calcium was 8.9 and the phosphorus 3.0 milligrams. The patient did not develop typical tetany. She spoke only of some slight numbness of the finger tips, which lasted 4 to 5 days following operation.

October 18, 1934, 3½ months following operation, the patient's general condition was excellent. She was free from the aches and pains of which she originally complained while the blood chemistry findings remained normal. The blood serum calcium was 9.7 and that of the phosphorus 3.6 milligrams. Patient was taking 1 heaping teaspoonful of calcium lactate by mouth three times a day. Otherwise, she occasionally experienced slight twitching sensations in her legs. The clinical signs of tetany were negative.

December 21, 1934, 5½ months following operation, the blood serum calcium was 9.6 milligrams and phosphorus 3.1 milligrams. The general condition was excellent. Patient felt very much better than she

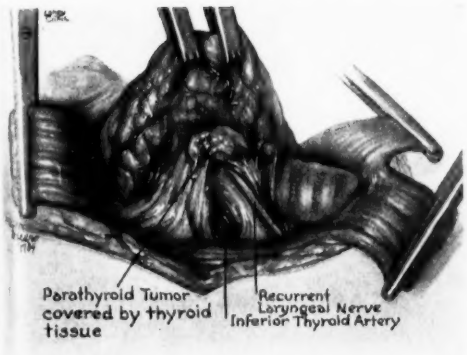

Fig. 30. Case 5. This parathyroid adenoma of the right inferior parathyroid was found at a location typical for inferior parathyroids at the point where they are most commonly seen in the course of thyroid operations.

The trunk of the inferior thyroid artery is shown and the course of the recurrent laryngeal nerve before it passes over or under the branches of the inferior thyroid artery. We have now dissected and visualized the recurrent laryngeal nerve several hundred times and are impressed with the fact that its course and location is dependably constant. Note that these tumors are not infrequently covered by a little thyroid tissue.

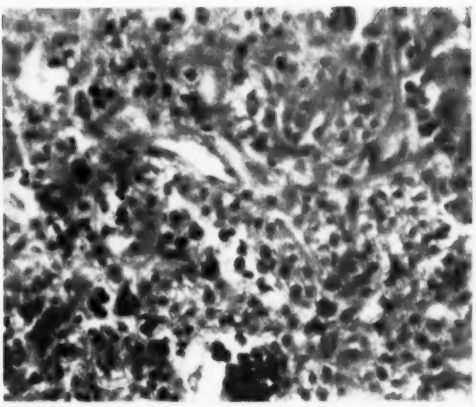

Fig. 31. Case 5. Photomicrograph of parathyroid adenoma removed from this patient. ×500.

had at any time during the past 2 years. Her only complaint was occasional backache in the region of the kyphos of the spine. These symptoms, however, were much less pronounced than they were before operation. She was continuing her calcium phosphate and lactate by mouth. No clinical signs or symptoms of tetany.

Case 5. Mrs. Maude F., aged 52 years, housewife. February 22, 1934, patient entered the clinic because of aching pains throughout the body and a severe pain localized in the mid low back and running around into the abdomen. X-rays of the spine revealed a generalized decalcification. There was definite compression of the body of the first lumbar vertebra (Fig. 29). It was thought that these findings would adequately explain the patient's pain, but in view of the decalcification of the bones, X-rays were also taken of the bones of the extremity and of the skull. The former were negative. The latter revealed a few mottled areas of irregular density with some actual thickening of the cortices. X-rays of the kidneys were negative for calculi.

The blood serum calcium ranged from 14.1 to 13.8 while the phosphorus varied from 2.1 to 2 milligrams. Phosphatase activity was 44.8 Bodansky units. The urine sediment revealed many white blood cells. The renal function test was normal.

Study of the X-rays of the spine first suggested that the compressed first lumbar vertebra might be the result of an old fracture, as there was definite increased density in the body of this bone. Paget's disease was considered. To rule out the possibility of any intraspinal pressure two lumbar punctures were performed with normal dynamics and with clear spinal fluid. The total spinal fluid protein was 50 milligrams. There was a cell count of 1, and a colloidal gold of 1,121,100,000. The calcium estimation in the spinal fluid was 6.0 milligrams.

In view of the laboratory findings, the fact that the patient was in negative calcium balance, the demonstration of a multiple colloid adenomatous goiter and the probability of a tumor of the parathyroid—it was decided to explore the thyroid gland, remove the adenomatous goiter, and search for a parathyroid adenoma. It was concluded that the patient presented strongly suggestive evidence of hyperparathyroidism, and that she also probably had a mild form of Paget's disease.

March 10, 1934, operation was done (FHL). An exposure of the thyroid region revealed a marble sized adenoma of thyroid tissue in the left lower lobe. At the right inferior pole, the typical position of a parathyroid body, a discrete tumor the size of a large lima bean was found in immediate relationship to the inferior thyroid artery and the recurrent nerve. The parathyroid adenoma shown in the artist's drawing (Fig. 30), made at the operating table, is about half the actual size. This mass was excised and on frozen section a diagnosis of parathyroid adenoma was made. This was later confirmed by complete histologic studies (Fig. 31).

Following operation the blood serum calcium was reduced to an average between 7.8 and 8.2 milligrams, while the phosphorus varied between 2.4 and 3.7 milligrams. Three days following operation the patient had a transitory attack of tetany with a positive Chvostek sign and numbness of the extremities. All symptoms of tetany were relieved by parathormone and by calcium lactate, grams 32 daily. She was discharged from the hospital 21 days after operation, entirely free from tetany. She was advised to continue 1 heaping teaspoonful of calcium lactate twice daily; 2 viosterol capsules, daily.

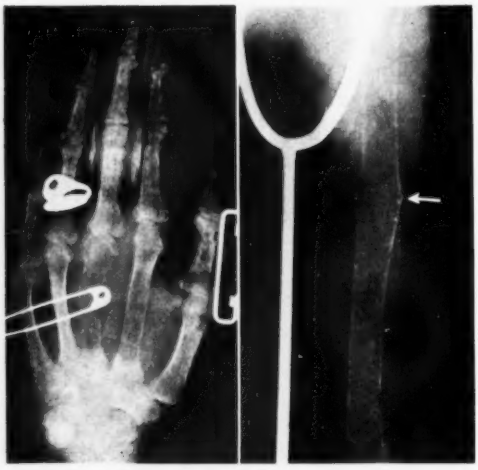

Fig. 32, a and b. An X-ray picture of the case of hyperparathyroidism seen in consultation with Dr. R. C. Cochrane. The two normal parathyroids had already been removed without relief of the hyperparathyroidism. At the suggestion of one of us (FHL), an intrathyroid parathyroid adenoma was found and removed at a third operation. It is shown to demonstrate the advanced state of decalcification in Figure 32a and the very slight reaction in the cortex in Figure 32b, the point of a spontaneous fracture.

Following operative convalescence progressive hyperextension of the spine was instituted to prevent further compression of the first lumbar vertebra. This treatment had to be discontinued because the patient would not co-operate. We were successful in securing the patient's agreement to wear a high supporting corset.

May 15, 1934, 2 months following operation the blood serum calcium was 10.1 milligrams and the phosphorus 4.9 milligrams, while the phosphatase activity was 35.0 Bodansky units. It was necessary to administer parathormone subcutaneously and calcium lactate by mouth to prevent constant tingling sensations and numbness in the extremities.

July 13, 1934, 4 months postoperative, this patient had gained 12 pounds in weight. Her chief complaint still was "kinking" in the low back. Blood serum calcium 11.2 milligrams, phosphorus 5.1 milligrams, and phosphatase 25.0 Bodansky units.

December 15, 1934, 8 months after operation, her general condition was still further improved. She had gained 10 pounds in weight. There were no clinical signs of tetany but she required calcium lactate (grains 45) daily, by mouth, to "feel good." The back pain referred to the lumbodorsal region was intermittently severe.

X-rays of the spine showed apparent normal calcification. The first lumbar body was compressed more than when first seen and was very dense. There was some early hypertrophic lipping of this vertebral body and of the bodies of the adjacent twelfth dorsal and second lumbar vertebræ.

Blood serum calcium was 9.8 milligrams; phosphorus, 3.5 milligrams; and phosphatase, 5.8 Bodansky units.

The back pain is now due entirely to the mechanical derangement of the lumbodorsal spine as a result of the compression of the body of the first lumbar vertebra. Early hypertrophic changes have also appeared. To date patient refuses treatment for this condition (Fig. 32).

The hyperparathyroidism is relieved.

SUMMARY

Five new proved cases of hyperparathyroidism due to parathyroid adenomata are recorded, together with the findings which lead to their diagnosis.

The clinical aspects, the blood chemistry, and the bone changes are discussed.

The appearance of normal and abnormal parathyroids is discussed. The normal and abnormal location of parathyroids is presented in graphic form.

The five proved cases of hyperparathyroidism together with X-rays of the bones in these cases, illustrations made at the operating table of the demonstration of the parathyroid tumors in such cases, and microscopic slides of the tumor are submitted.

We must all, particularly the orthopedist and the urologist, be on the lookout for hyperparathyroidism in patients complaining of pain in the back or extremities, in patients with diffuse neuritis or arthritic pains made worse on motion, in patients with progressive loss in stature, and in patients with kidney stones. Many patients with hyperparathyroidism who could be relieved of their condition by surgical removal of a parathyroid adenoma, have in the past and are doubtless still passing through our hands with their condition unrecognized.

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MECHANISM, SYMPTOMS, AND TREATMENT OF HERNIA INTO THE DESCENDING MESOCOLON (LEFT DUODENAL HERNIA)

A Plea for a Change in Nomenclature[1]

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IT is our purpose in this paper to elaborate upon several phases of the so called "left duodenal" variety of intraperitoneal (internal) hernia. First, we shall report a case of a complete rupture of this type; an attempt will be made to explain the hernia on an embryologic basis; reasons will be adduced to justify a change in the nomenclature of this lesion. The symptoms and signs consequent upon strangulation of the hernial contents, and the methods of treatment of this variety of hernia will be mentioned.

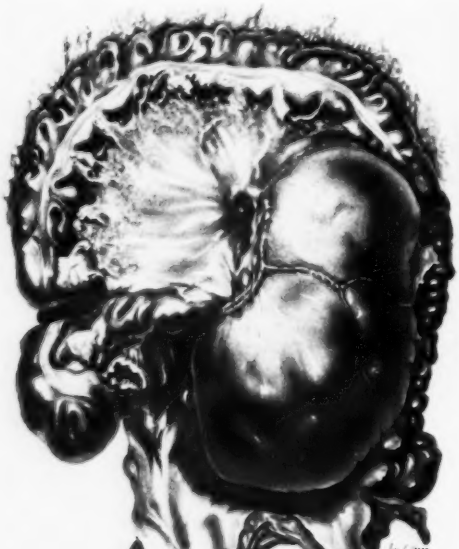

Fig. 1. Appearance of hernial mass of jejuno-ileum. The topography of the colon is normal. All but about 5 centimeters of the jejuno-ileum is incarcerated in the sac, derived from descending mesocolon. In this drawing the neck of the sac is shown as open; blunt and sharp dissection was required to demonstrate the opening. The ascending branch of the inferior mesenteric artery and the inferior mesenteric vein are in the anterior wall of the neck of sac.

REPORT OF CASE

A male, E.S., 40 years of age, was admitted to the Mount Zion hospital because of rheumatic heart disease. His death in the hospital was the result of chronic mitral valvulitis, obliterative pericarditis, and lobar pneumonia. In the course of his hospitalization he gave no history and presented neither signs nor symptoms of the unusual intraperitoneal hernia which he presented at autopsy examination.

Our interest in the postmortem examination centered about an enormous hernial sac (Fig. 1) within which practically all of the jejuno-ileum was incarcerated. When the abdominal cavity was opened, the stomach, duodenum, liver, and spleen were in their proper positions and had maintained their normal relationships. The cæcum and the ascending, transverse, and descending divisions of the colon were in the normal inverted U position. The ascending and descending mesocolon and the segments of large bowel attached to the mesocolon were applied and normally adherent to the posterior parietal

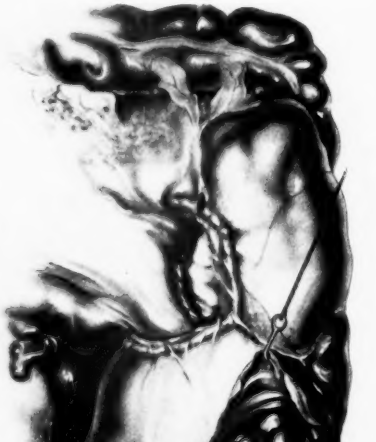

Fig. 2. An incision was made in the peritoneal layers, in an avascular area, in the anterior wall of the hernial sac. The jejuno-ileum is seen through the rent in the sac.

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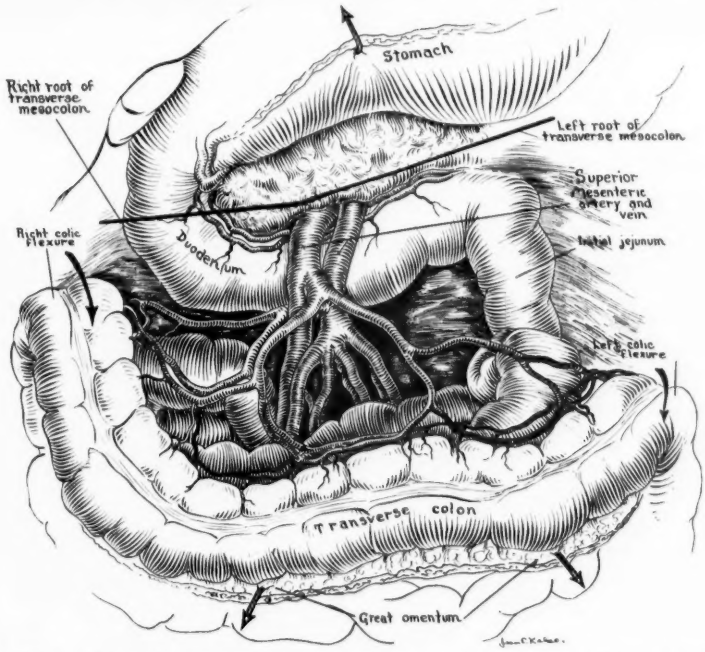

Fig. 3. Dissection in recorded case of region about the transverse division of the duodenum and the initial jejunum. This drawing was made to show that the duodenum lay behind the superior mesenteric vessels. The colic angles are depressed to allow illustration of the distribution of the superior mesenteric vessels to the ascending and transverse colons.

peritoneum. The sigmoid colon was omega in shape and was suspended by a mobile mesentery.

Only the terminal 5 centimeters of the small bowel could be seen; the remainder of the jejuno-ileum lay within a hernial sac produced at the expense of the descending mesocolon, and formed a large left sided abdominal tumor. The forward wall of the hernial sac was the descending mesocolon, the serosal duplication of which contained the ascending branch of the inferior mesenteric artery and the inferior mesenteric vein, both of which stood out prominently. The location of the neck of the sac at first was not apparent for the reason that the margins of the orifice were united by peritoneal "agglutination." However, blunt dissection about the duodenojejunal angle disclosed the neck of the sac which was a palm's breadth in length, and demonstrated its several margins. There was no evidence of periduodenal folds or fossæ. In the forward margin of the sac ran the ascending branch of the inferior mesenteric artery and the main trunk of the inferior mesenteric vein. In the inferior limit of the neck of the sac was the concavity of the upwardly arching course of the ascending branch of the inferior mesenteric artery. The superior boundary of the neck of the sac was the inferior mesenteric vein which ran in a medial direction to contribute to the formation of the portal vein. The posterior boundary of the neck of the sac was the primitive midline attachment of the descending mesocolon.

An incision was made through the peritoneal layers which form the anterior wall of the sac, i.e., through an avascular area of the descending mesocolon, and the jejuno-ileal contents of the sac were exposed (Fig. 2). Had strangulation taken place within the sac, an incision so placed as to avoid injury to the blood supply of the descending colon would have been used to determine the cause of obstruction and to restore alimentary continuity. Figure 4 shows all of the small bowel exteriorized through a large opening in the anterior wall of the sac. There were no adhesions between the contents of the sac and the sac wall.

The contents of the sac were then replaced and the intestinal coils were drawn to the right through the neck of the sac into the general abdominal cavity as is illustrated in Figure 5. It is evident that the root of the mesentery of the small bowel is much shorter than that found in the normal abdominal cavity. The drawing indicates the degree to which the inferior or distal coils of small bowel are bunched or clumped. The anatomical explanation for the

Fig. 4. All of the jejuno-ileum has been withdrawn through the rent in the anterior wall of the hernial sac. There are no adhesions between the hernial contents save at the neck of the sac.

Fig. 5. Mass of jejuno-ileum withdrawn through neck of sac into general abdominal cavity. The rent in the anterior wall of the sac noted in the foregoing drawings is retracted with hooks. The dissected area shows the upward course of the ascending branch of the inferior mesenteric artery and the inferior mesenteric vein. Both vascular trunks run in the anterior wall of the neck of the sac and constrict the sac orifice.

clumping of the distal loops of small bowel lies in the overlapping of the small bowel mesentery in its terminal portion. The mesentery at and near its root is compressed and partly turned upon itself to such a degree that two-thirds of the small bowel seem attached to that part of the mesentery which apparently has no more than 5 centimeters of posterior attachment.

Attempts made to separate the layers of peritoneum which form the walls of the sac were unsuccessful in demonstrating how many primitive layers were present before embryologic coalescence of opposed serous surfaces took place. A determination of the number of primitive peritoneal layers involved also was attempted by sectioning tissue blocks of the sac walls. The formation of coalescent fusion fascias rendered layer differentiation impossible.

GASTRO-INTESTINAL EMBRYOLOGIC BACKGROUND FOR INTERPRETATION OF THIS HERNIA

Before attempting to explain the variety of mechanisms by which this intraperitoneal hernia occurs, it is necessary to review the essential stages in the development of the gastro-intestinal tract and its associated peritoneum.

The early alimentary canal traverses the future abdominal cavity of the embryo as a straight tube suspended from the dorsal wall by a common dorsal mesentery (Fig. 6). The tube is suspended anteriorly over the superior portion of its extent by the ventral common mesentery. As compared with the gradual growth of the embryo in length, disproportionately large growth occurs in the primitive digestive tube—one which results in the segmentation of the tube into its ultimate constituents, the stomach and the small and large bowel.

Differentiation of the tube results in a differentiation within its carrying mechanism, the dorsal and ventral mesenteries. These are subdivided into the dorsal and ventral mesogastria, the mesoduodenum, and the mesenteries of the small and large bowel. The distribution of the branches of the aorta to each segment of the alimentary tube takes place

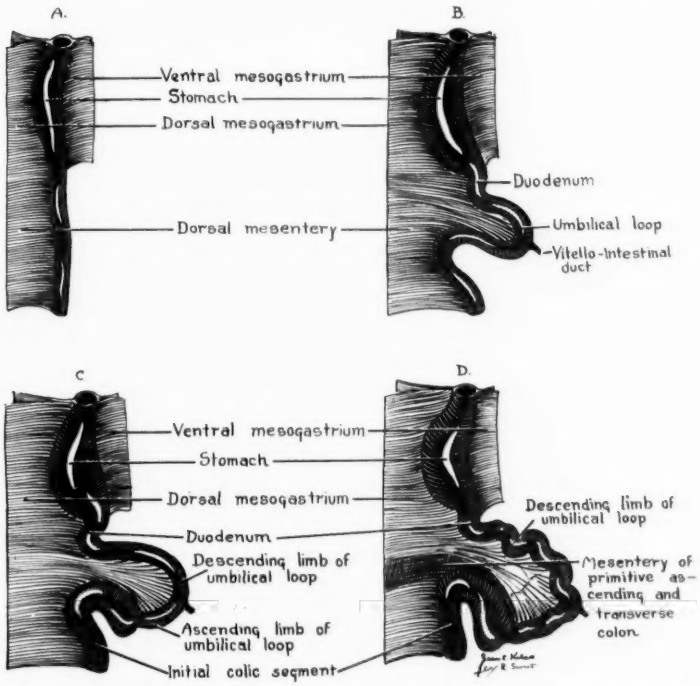

Fig. 6. Development of the gastro-intestinal tract from the primitive sagittal alimentary tube to the umbilical loop stage. A, The primitive stomach is shown as a fusiform swelling with its greater curvature directed dorsally. The ventral mesentery extends a little beyond the stomach. B, The gastric curvatures are more differentiated. The umbilical loop is suggested. The mesentery of the loop hypertrophies to accommodate the increase in length of the intestinal tube which forms the loop. C, The ascending limb of the loop, at the point which marks the ileocæcal junction, enlarges and forms the cæcum, or beginning of the initial segment of the colon. D, The small intestine segment of the loop (jejuno-ileum) increases in length, as shown by the fluting. The differentiation of the dorsal common mesentery into segments is indicated.

while the tube and mesenteries are in the primitive sagittal position.

The structural changes in the tube are not all those of simple elongation, for there are changes in form and capacity as well. The cephalic portion of the canal bulges into a fusiform swelling, the stomach. The middle portion lengthens into the redundant umbilical loop, of which the descending limb and part of the ascending limb constitute small intestine, and the remainder of the ascending limb becomes the initial part of the large bowel. The terminal part of the alimentary tube is transformed into a long, uniformly large segment, the remainder of the large bowel.

While these changes occur, the glands adnexed to the digestive tract develop between the peritoneal duplications which make up the dorsal and ventral mesenteries; these are the liver, spleen, and pancreas. The spleen develops independently of the gastro-intestinal tube, but the liver and pancreas arise as outgrowths from it (Fig. 7).

The rotation and posterior fixation of the gastroduodenal segment of the alimentary tract will not be discussed at length because the changes which take place in this segment have no bearing upon the mechanism of left duodenal hernia. The stomach in this rotation is the central and pivotal viscus. The first rotation of the stomach is about its long

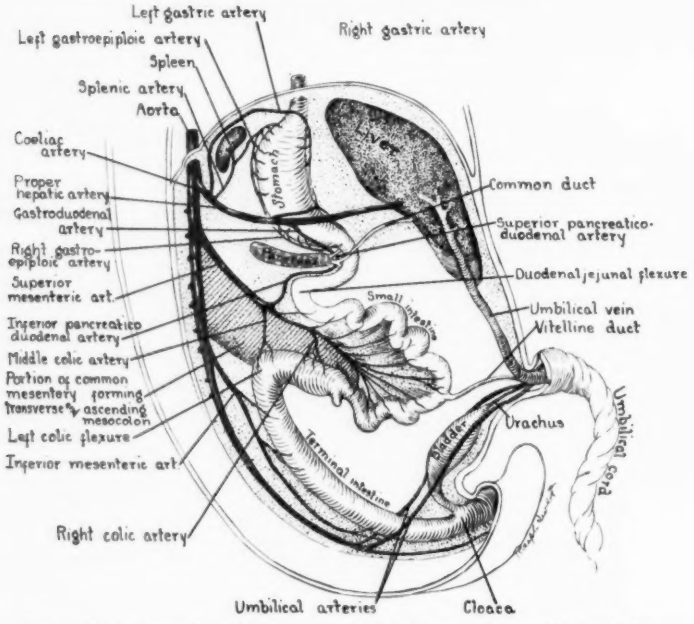

Fig. 7. Diagrammatic sagittal section of an embryo at the completion of the umbilical loop stage of development. The cœliac artery supplies the gastroduodenal segment, the superior mesenteric artery the umbilical loop, and the inferior mesenteric artery supplies the terminal intestine. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

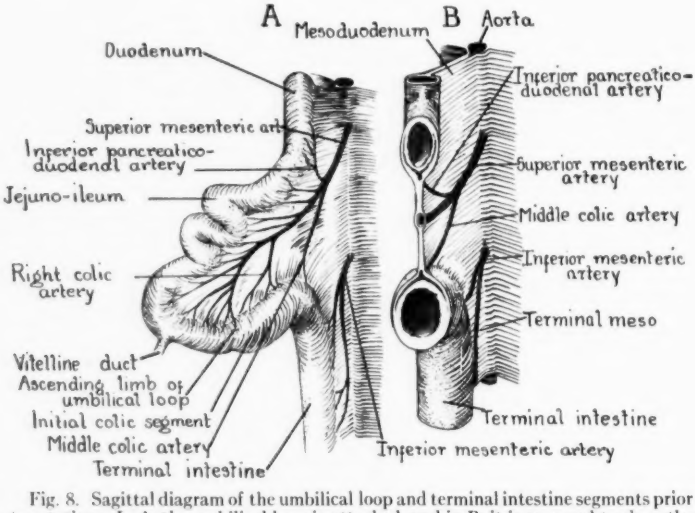

Fig. 8. Sagittal diagram of the umbilical loop and terminal intestine segments prior to rotation. In A, the umbilical loop is attached, and in B, it is removed to show the distribution of the superior mesenteric artery. The superior mesenteric artery is the axis of the loop, about which rotation occurs. The inferior mesenteric artery is the vascular supply of the terminal intestine. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

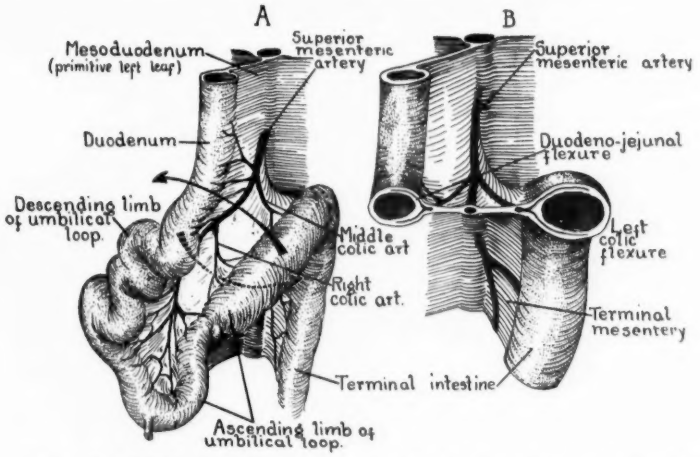

Fig. 9. Umbilical loop segment after a counter clockwise rotation of 90 degrees. In A, the loop maintains its duodenal and terminal colon connections. In B, the ascending and descending limbs of the loop are removed to show the disposition of the mesentery and the enclosed arterial supply. The arrow indicates the direction of the succeeding stage of rotation. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

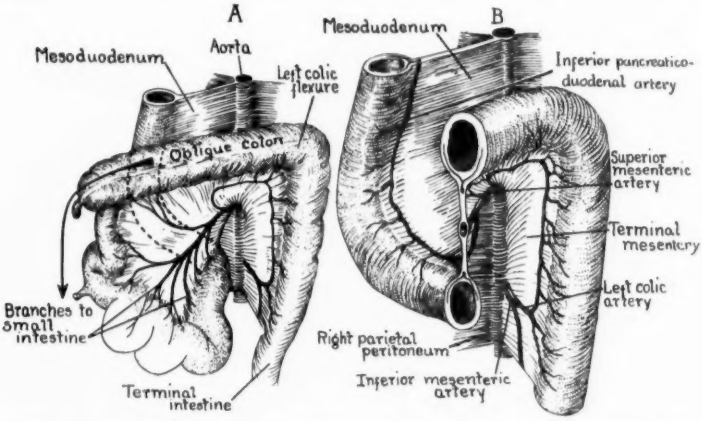

Fig. 10. Disposition of the structures formed from the umbilical loop after its rotation to the oblique colon stage. In A, the elements of both limbs of the loop maintain their duodenal and terminal colon connections. In B, the elements of both limbs of the loop are removed. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

axis and the second rotation about its anteroposterior axis. The rotation of the stomach upon its anteroposterior axis folds the initial part of the primitively sagittal duodenum and its mesentery and contents against the right primitive parietal peritoneum, to which they become fused by the process of fetal peritoneal "agglutination."

An understanding of the rotation and posterior fixation of the umbilical loop segment of the gastro-intestinal tract is essential to the comprehension of the mechanism of left duodenal hernia. We have referred to the initial part of the duodenum as having been brought to the right across the median line by the rotation of the stomach about its antero-

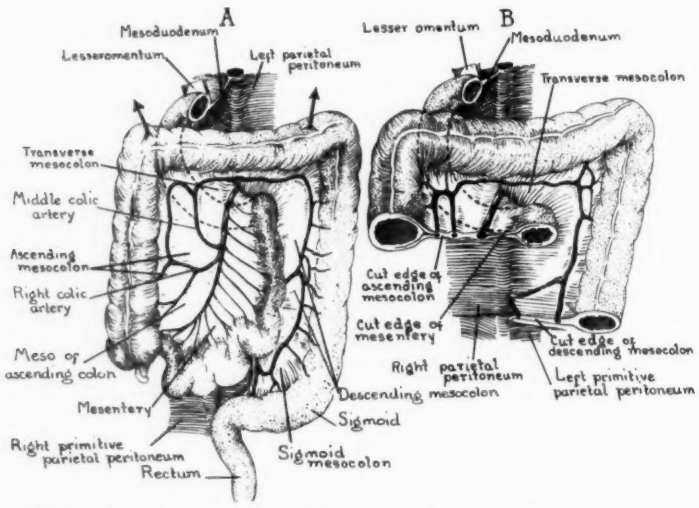

Fig. 11. Final stage of umbilical loop rotation. In A, the intestinal structures are in place after the completion of rotation and the formation of the ascending and transverse colon segments from the oblique colon. In B, most of the ascending colon and mesocolon and the jejuno-ileum and its mesentery are removed. The superior artery is the boundary between the mesentery of the ascending colon and the mesentery of the jejuno-ileum. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

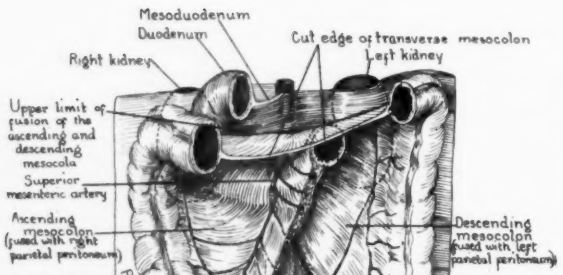
Mesoduodenum
Duodenum
Cut edge of transverse mesocolon
Right kidney
Left kidney
Upper limit of fusion of the ascending and descending mesocola
Superior mesenteric artery
Ascending mesocolon (fused with right parietal peritoneum)
Descending mesocolon (fused with left parietal peritoneum)
Right parietal peritoneum
Left parietal peritoneum
Cut edge of mesentery (mobile as far as superior mesenteric artery)

Fig. 12. Diagram to show the mesenteries of the ascending, transverse, and descending portions of the colon in their ultimate positions prior to posterior peritoneal fixation. The jejuno-ileum is cut away to show its mesentery which remains mobile. The root of the mesentery is along the line of the superior mesenteric artery. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

posterior axis. The distal part of the duodenum is the connection between the gastroduodenal segment and the umbilical loop. The colic angle or the inferior connection of the loop is the junction between the distal end of the umbilical loop and the terminal intestine. The ultimate position of the terminal part of the duodenum is dependent upon the rotational changes of the umbilical loop, since these changes affect the position of the superior and inferior connections or points of suspension of the loop, the former of which includes the terminal duodenum.

In order to make clear the various changes in position undergone by the umbilical loop, it will be helpful to follow these changes in position as illustrated in Figures 8 to 11 inclusive, as though one were producing them manually.

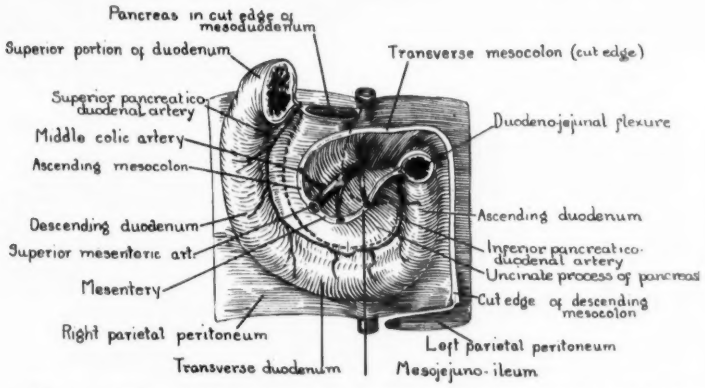

Fig. 13. Diagram of the duodenum and mesoduodenum after rotation of the gastro-duodenal and umbilical loop segments of the alimentary tube. The diagram shows how these structures are applied and fixed to the underlying peritoneum; the descending duodenum and part of the transverse duodenum are applied to and fuse with the primitive right parietal peritoneum. The remainder of the transverse duodenum and the ascending duodenum are applied to and fuse with the descending mesocolon. The duodenojejunal angle fuses partly with the inferior part of the transverse mesocolon. (Callander's *Surgical Anatomy*. Courtesy W. B. Saunders Co.)

After rotation is complete, the ascending, transverse, and descending portions of the colon are illustrated as having been carried to their ultimate position of an inverted U, with the loops of the jejunum and ileum surrounded by the large bowel as by a frame. The key to the permanent and fixed arrangement of the segments of the gastro-intestinal tract lies in the manner of their posterior peritoneal fixation. The cæcum, ascending colon, and the ascending mesocolon fuse with the right primitive parietal peritoneum over that area included between the trunk of the superior mesenteric artery and the lateral margins of the ascending colon. The transverse colon and mesocolon do not fuse with the primitive posterior parietal peritoneum but make up a more or less mobile unit. Posterior peritoneal agglutination binds the descending colon and mesocolon to the left primitive parietal peritoneum.

Our interest in recording the steps in gastro-intestinal fixation centers about the terminal duodenum. It will be noted by referring to Figure 13 that the terminal duodenum and its mesentery and the continuous mesentery of the transverse and descending colon are superimposed structures before peritoneal agglutination takes place. The descending mesocolon fuses with the left parietal peritoneum, and the ascending duodenum and its mesentery and the duodenojejunal angle fuse to the underlying mesocolon. The degree of fixation of the terminal duodenum to the mesocolon, whether hypofixation or hyperfixation, determines the strength of adhesions which bind down this segment of bowel to the mesocolon. If the adhesion of the duodenum to the mesocolon is strong, the superior and inferior duodenal (duodenojejunal) folds are shortened and the recesses they roof (superior and inferior duodenal fossæ) are shallow and small. If, on the other hand, the adhesion of the duodenum and its mesentery to its mesocolon is loose, then these folds are large, and the fossæ which they roof are broad and deep.

THE FOSSÆ ABOUT THE TERMINAL DUODENUM

In an analysis of the mechanism of hernia into the descending mesocolon, a detailed description of the recesses about the terminal duodenum must be given because one or several of these fossæ undoubtedly may be the point of entrance of small bowel in the development of this type of rupture. The most important of these recesses are the superior

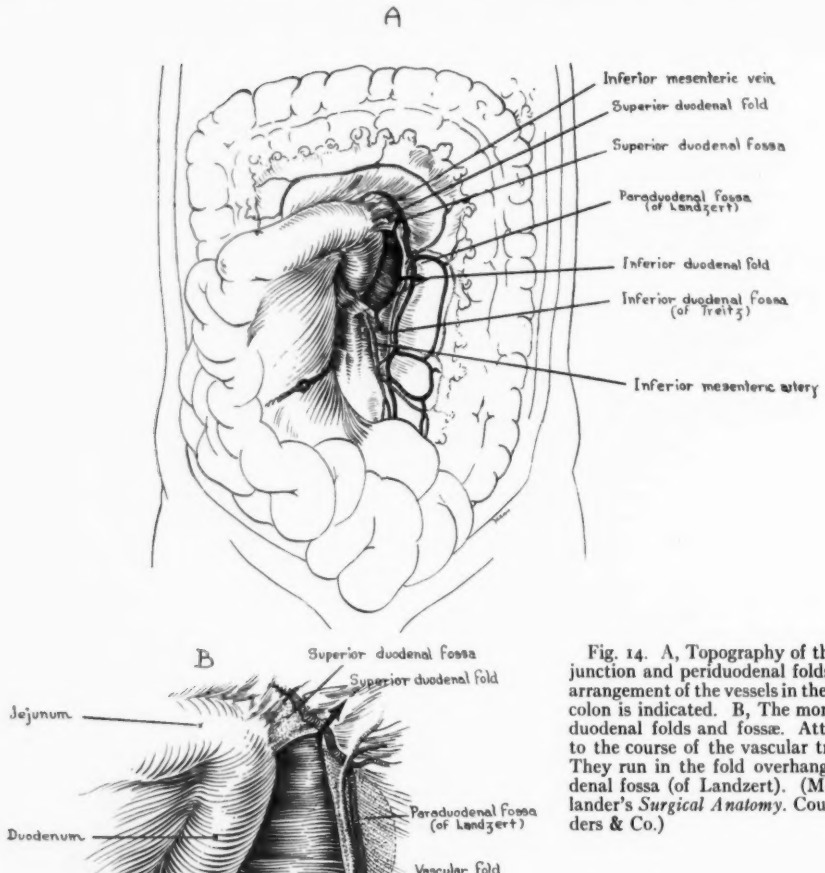

Fig. 14. A, Topography of the duodenojejunal junction and periduodenal folds and fossæ. The arrangement of the vessels in the descending mesocolon is indicated. B, The more important periduodenal folds and fossæ. Attention is directed to the course of the vascular trunks noted in A. They run in the fold overhanging the paraduodenal fossa (of Landzert). (Modified from Callander's *Surgical Anatomy*. Courtesy W. B. Saunders & Co.)

duodenal, inferior duodenal, and the paraduodenal fossæ.

The superior duodenal fossa is the most constant of the peritoneal depressions situated about the ascending portion of the duodenum and to the left of the termination of this segment of the small bowel. The fossa lies at the level of the second lumbar vertebra and is bounded anteriorly by the superior duodenal fold, a duplication of peritoneum which is essentially an embryological adhesion between the termination of the duodenum and the inferior surface of the transverse mesocolon near its union to the descending mesocolon. The inferior, or free, margin of the fold is concave, its mesial extremity blending with the peritoneum over the anterior surface of the terminal duodenum, and its lateral extremity

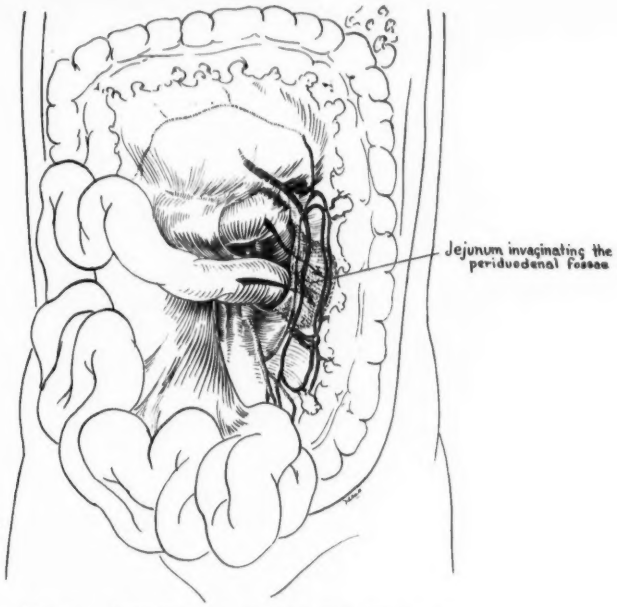

Fig. 15. Invagination of periduodenal fossæ and, later, of descending mesocolon by initial knuckle of jejunum. The invagination is mainly into the paraduodenal fossa (of Landzert).

fusing with the ultimate parietal peritoneum at the junction of the transverse and descending mesocolon, i.e., anterior to the left kidney. The lateral attachment of the superior duodenal fold may be mesial or just lateral to the course of the inferior mesenteric vein; hence, the designation of this fold as a "vascular plica." The orifice of the superior duodenal fossa looks inferiorly and its apex extends superiorly, sometimes as far as the body of the pancreas.

The inferior duodenal fossa (of Treitz) lies to the left of the ascending portion of the duodenum on a level with the third lumbar vertebra. Its orifice looks superiorly directly into the orifice of the superior duodenal fossa. The closed end of the fossa points inferiorly and to the right toward the mesentery of the proximal jejunum. The cavity is bounded anteriorly by the inferior duodenal fold, the free margin of which is thin and sharply demarcated. Its medial extremity blends with the peritoneum over the anterior surface of the ascending duodenum, and its lateral

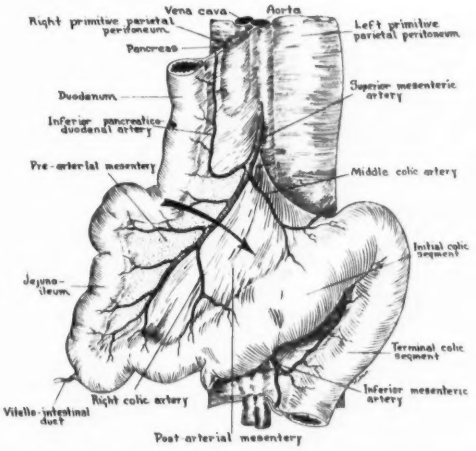

Fig. 16. Position of the small and large bowel at end of first stage of rotation. The arrow indicates the direction taken by the jejuno-ileum in invaginating the postarterial mesentery (ultimate ascending and transverse mesocolon). This drawing is explanatory of Haymond and Dragstedt's case of anomalous rotations. This and the following drawings were made as modifications of their drawings in an attempt to illustrate in 3 dimensions the anomaly they report.

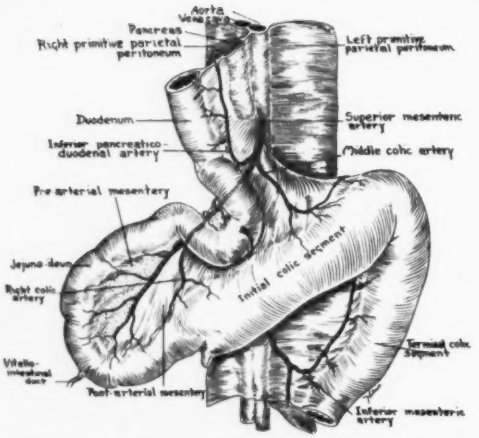

Fig. 17. Invagination of the postarterial mesentery by the small bowel. The duodenum, instead of rotating counter-clockwise behind the superior mesenteric artery, crosses anterior to the artery and is beginning to invaginate the postarterial mesentery (ultimate ascending and transverse mesocolon). The initial colic segment ultimately will become the ascending and transverse divisions of the colon.

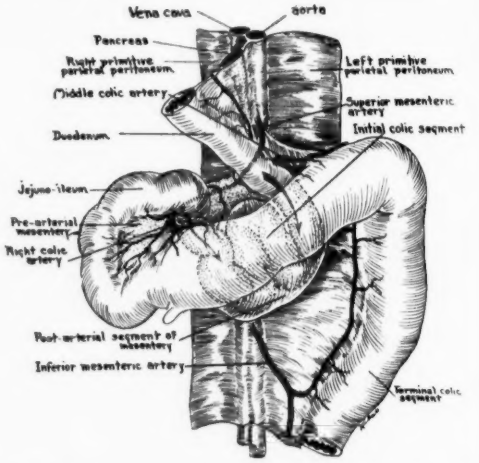

Fig. 18. Invagination of the postarterial mesentery by the small bowel. Much of the duodenum and jejuno-ileum now has pocketed itself into the postarterial mesentery. The oblique colon as yet has not become differentiated into the ascending and transverse colon.

extremity fuses with the peritoneum of the descending mesocolon (ultimate posterior parietal peritoneum). The inferior mesenteric vein and left colic artery usually lie well to the left of the lateral attachment of the inferior duodenal fold, although occasionally this vein has been found in the posterior wall of the fossa under discussion. The inferior duodenal fossa usually can accommodate the first phalanx

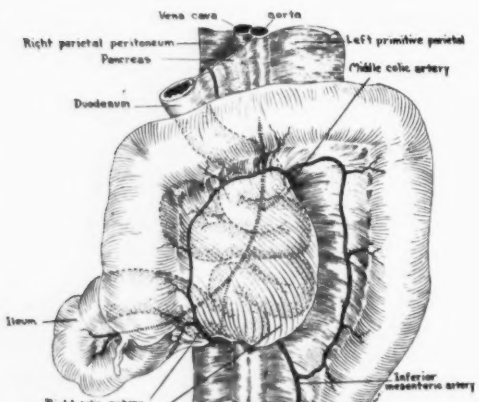

Fig. 19. Invagination of the postarterial mesentery by the small bowel. The mass of small bowel continues to invaginate the postarterial mesentery. The initial colic segment now rotates and elongates until the ascending and descending divisions of the colon are visualized.

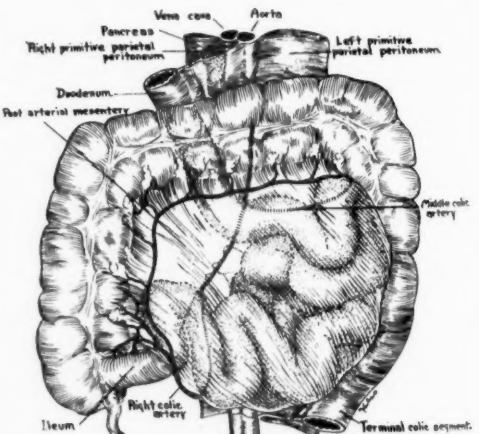

Fig. 20. Invagination of the postarterial mesentery by the small bowel. The divisions of the colon now are in their normal positions. The mass of jejuno-ileum is contained in a sac derived from postarterial mesentery. Most of the sac and contents lie to the left of the midline. Both the duodenum and terminal ileum cross anterior to the superior mesenteric artery. This schema represents the final stage in the production of the anomaly observed by Haymond and Dragstedt.

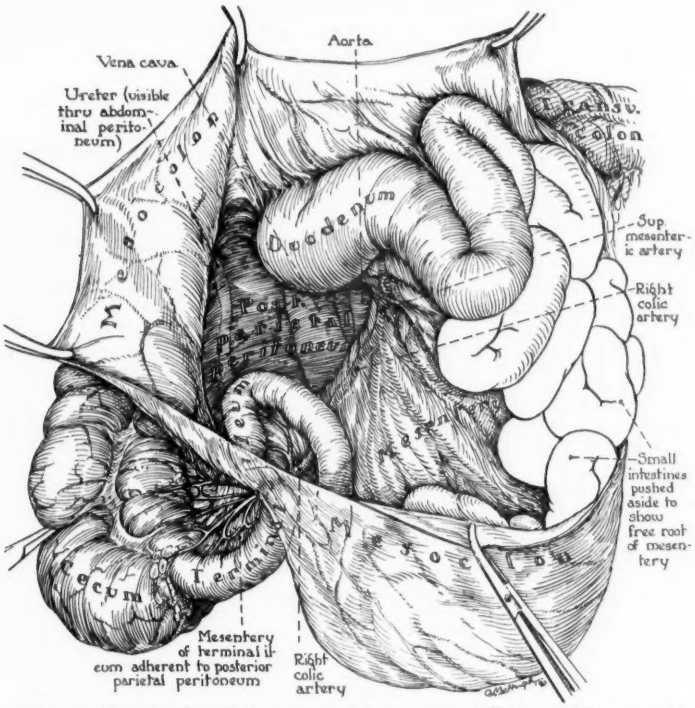
Aorta
Vena cava
Ureter (visible thru abdominal peritoneum)
Duodenum
Sup. mesenteric artery
Right colic artery
Small intestines pushed aside to show free root of mesentery
Mesentery of terminal ileum adherent to posterior parietal peritoneum
Right colic artery

Fig. 21. Invagination of the postarterial mesentery by the small bowel. This drawing is a photograph of Haymond and Dragstedt's anomaly. Incision of the postarterial mesentery (ascending and transverse mesocolon) shows the contents of the sac at autopsy. It is obvious that the duodenum and the terminal ileum run anterior to the superior mesenteric artery. (From Haymond and Dragstedt, *Surg., Gynec. & Obst.*, 1931, 53:323.)

of the middle finger. Occasionally, the fossa extends into the region posterior to the ascending portion of the duodenum, a result of inadequate peritoneal fixation of this portion of the bowel.

The paraduodenal fossa (of Landzert) frequently exists in conjunction with the two fossæ just described. It is located at the left and quite a distance from the ascending portion of the duodenum, and is a cavity produced by the elevation of a fold of peritoneum by the inferior mesenteric vein. This fold has been aptly termed the fold of the inferior mesenteric vein in its course within the descending mesocolon; hence, the varying distance from the terminal duodenum of the peritoneal fold which contains this vessel. The floor of the paraduodenal fossa is the peritoneum of the descending mesocolon (ultimate posterior parietal peritoneum). The mouth or neck of the fossa is wide and opens inferiorly and to the right, and its closed extremity is directed superiorly and to the left. When the paraduodenal fossa occurs in conjunction with the superior or the inferior duodenal fossa, or with both, there usually is an amalgamation of the folds roofing the orifices of the fossæ.

The posterior or retroduodenal fossa occurs occasionally, especially in conjunction with a large paraduodenal fossa. The posterior duodenal fossa lies immediately behind the superior part of the ascending division of the duodenum, and is the result of a lack of fusion between the terminal part of the duodenum and the underlying ultimate posterior parietal peritoneum (primitive descending mesocolon).

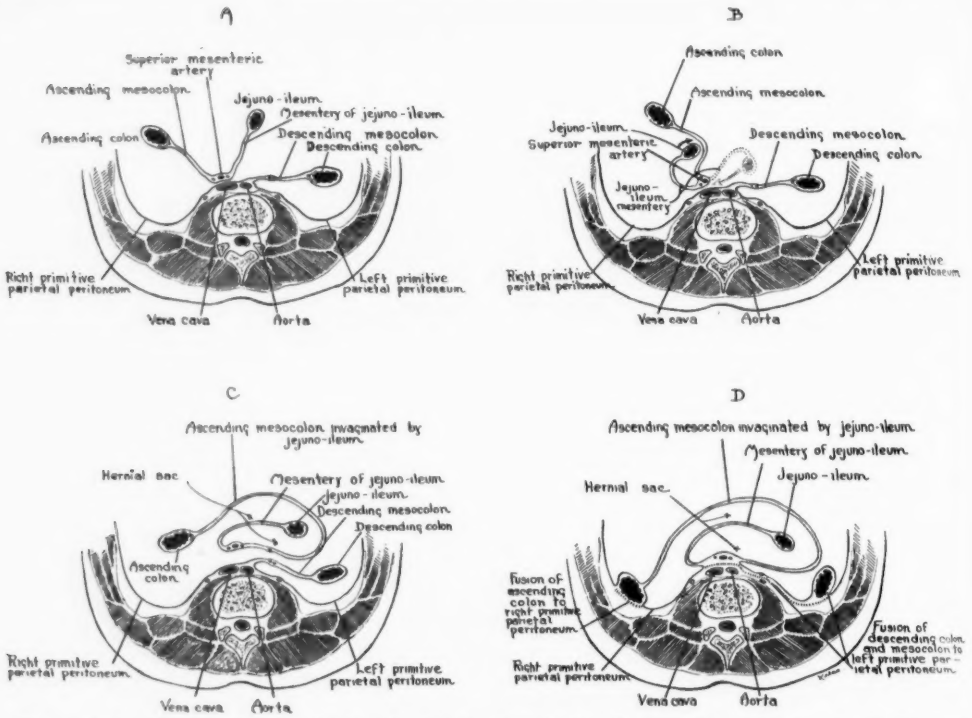

Fig. 22. Series of cross sections showing invagination of the postarterial mesentery by the small bowel. A, indicates the level at which invagination occurs. The jejuno-ileum and ascending colon are on a continuous mesentery. The ascending and descending mesocolons as yet are not fused to the posterior parietal peritoneum. The jejuno-ileum occupies its normal position after proper rotation has taken place. In B, the small bowel is illustrated as not having gained its normal position, as in A, by rotating behind the superior mesenteric artery, but by rotating clockwise and invaginating the ascending mesocolon. In C, the small bowel continues to invaginate and make a hernial sac of the ascending mesocolon. The ascending colon is dropping down, preparatory to fusing with the right primitive parietal peritoneum. In D, the ascending colon has fused with the right primitive parietal peritoneum. The descending colon and the mesocolon have fused with the left primitive parietal peritoneum. The small bowel is enclosed in a hernial sac which is comprised of the ascending mesocolon.

There can be little doubt that the peritoneal duplications which delimit the periduodenal fossa are fusion or adhesion folds which are the result of fetal agglutination or fixation. We have noted in the section devoted to the development of the gastro-intestinal tract how the terminal duodenum is rotated against, and becomes adherent to, the free peritoneal surface of the transverse and descending mesocolon. Hyperfixation, which results in strong agglutination of the opposed surfaces, makes for short folds and shallow fossæ. Hypofixation results in longer folds and deeper fossæ. Adhesions have been noted in the embryo in this region at the middle of the fourth fetal month. At this early stage, these adhesions are delicate and non-resistant. In embryos of greater age, the adhesions are denser and are capable of withstanding considerable tension.

THEORIES OF HERNIA DEVELOPMENT

Accredited mechanism of hernia development. As a result of the fundamental work by Treitz, Moynihan, Landzert, Broesike, and others, the genesis of the so called left duodenal type of hernia has been associated with the periduodenal fossæ. Almost all investigators of the lesion agree that it usually begins in early fetal life. The initial portion of the jejunum is assumed to be invaginated into

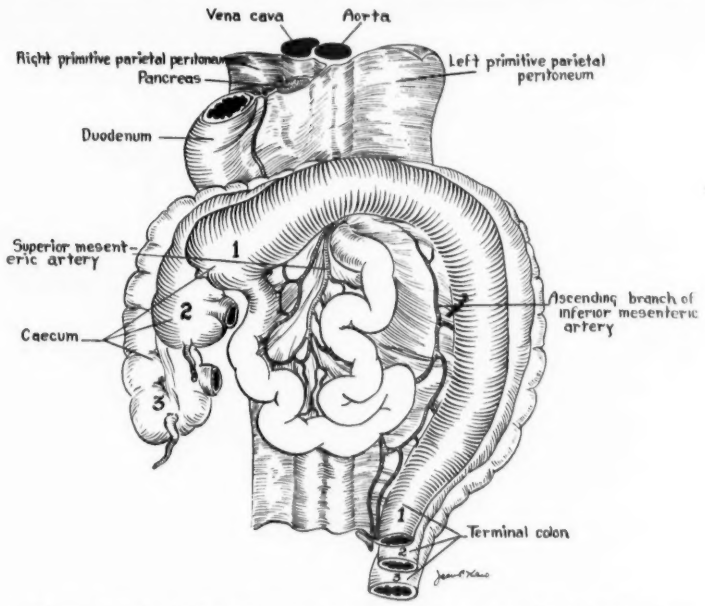

Fig. 23. Drawing indicating successive positions of large bowel and the mesocolon. The numbers indicate the stages in the progressive increase in size and in the placement of the large bowel and mesocolon.

the paraduodenal fossa (of Landzert) or into the inferior duodenal fossa (of Treitz) or into a combined fossa (of Treitz and Landzert). By a mechanism altogether obscure, the bowel gradually enlarges the fossal sac until a smaller or greater part of the jejuno-ileum is invaginated and lost to view.

That the periduodenal fossæ with their overhanging folds do offer areas of easy engagement for the small bowel is incontrovertible; it seems certain that many small ruptures of the type of hernia under discussion occur in which the periduodenal folds and fossæ are clearly delimited and the fossæ are occupied by one or more knuckles of small bowel. It is to be expected that the anterior part of the neck of the sac contain one or more of the constricting trunks of the inferior mesenteric vein and the ascending branch of the left colic artery.

It is still a matter of conjecture whether the hernia develops altogether *in utero*, or whether the enlargement of the sac continues after birth. It seems reasonable to assume that the sac reaches its maximum size in fetal life, since during part of that period the descending colon is attached to a mesocolon, sufficiently mobile to be invaginated easily and to contribute to the formation of a sac. There are those, however, who insist that the enormous sac required to house virtually all the jejuno-ileum may be formed after the primitive mobile descending mesocolon has been fixed to the primitive left parietal peritoneum.

In assuming that one of the periduodenal fossæ, particularly the paraduodenal fossa, is the point of origin of this rupture, and that these fossæ are depressions in the descending mesocolon, it is necessary also to assume that this increase in the size of the sac must be at the expense of the descending mesocolon. This must be by the progressive process of invagination, irrespective of the size which the hernia attains.

Invagination of the combined ascending and transverse mesocolon (post-arterial mesentery) by the small bowel. Haymond and Dragstedt have described a most unusual type of intra-

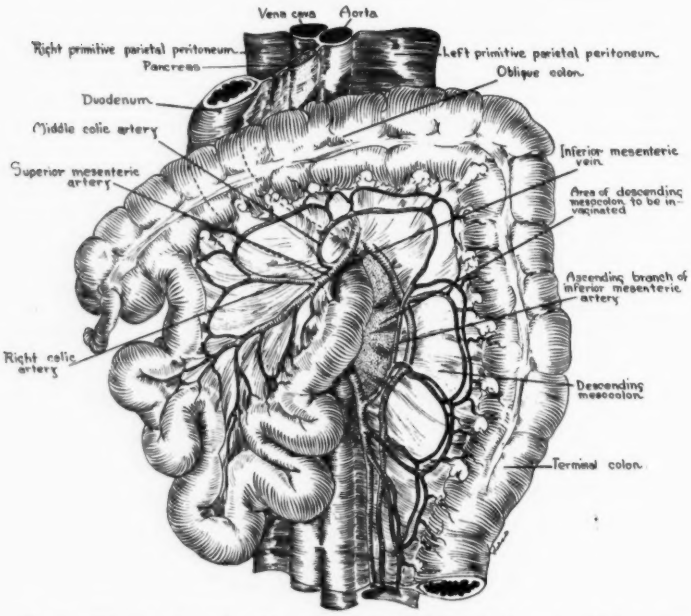

Fig. 24. Diagram of small and large bowel and their attached mesenteries in a stage of almost complete rotation. Posterior peritoneal fixation has not occurred as yet. The stippled area in the continuous transverse and descending mesocolons indicates the avascular area invaginated, according to our hypothesis, by the coils of jejuno-ileum before posterior peritoneal fixation has taken place.

peritoneal hernia. Their hernia is the result of an anomaly in the second stage of gastro-intestinal rotation in which the jejuno-ileum comes to occupy an abnormal position by invaginating the mesentery of the primitive ascending and transverse colon. We have modified certain drawings of these authors and have regrouped them in an attempt to make more clear the mechanism of the hernia they describe.

Figure 16 illustrates the midgut at the end of the first stage of rotation when the mass of jejuno-ileum is in the right side of the abdomen and the segments of large bowel are in the left. Figure 17 indicates that the jejuno-ileum, instead of rotating counter-clockwise about the superior mesenteric artery as an axis, rotates clockwise and invaginates the primitive mesentery of the ascending and transverse colon (post-arterial mesentery). When all the small bowel has invaginated this mesentery (Figs. 20 and 21), the duodenum of necessity crosses anterior to the trunk of the superior mesenteric artery and not posterior to it, as would be the case were small bowel rotation normal and therefore counter-clockwise (Fig. 10). The drawings (Figs. 16 to 23) indicate the manner in which the small bowel invaginates the post-arterial mesentery until all of the small bowel is in the mesentery as if in a sac.

No case of hernia into the descending mesocolon possibly can arise in this manner because the sac formed by the ascending mesocolon separates the hernial contents from any contact with the descending mesocolon (Figs. 17 to 21). The descending colon and descending mesocolon in the instance of malrotation under discussion are entirely normal in development, rotation, and posterior fixation.

A series of cross sections through this anomaly (Fig. 22) indicates how much this condition differs from that of hernia into the descending mesocolon (Fig. 27).

Proposed mechanism of development of hernia into the descending mesocolon. In the

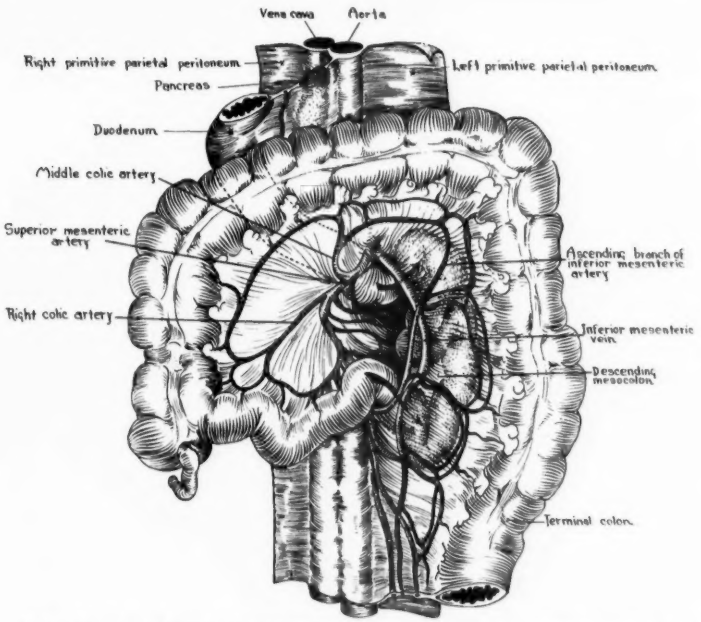

Fig. 25. Well advanced invagination of the as yet mobile descending mesocolon by coils of jejuno-ileum. It is evident that the sac is formed at the expense of the avascular area of descending mesocolon. Attention is drawn to the vascular trunks in the anterior wall of the neck of the sac.

development of a large hernia such as we are reporting, we believe that a mechanism can be postulated which does not presuppose that the small bowel invaginate the periduodenal fossæ, a fanciful theory in large ruptures. It seems logical that the loops of jejuno-ileum, as they pass normally behind the superior mesenteric artery as in Figure 24 to Figure 26, invaginate the mobile descending mesocolon in its upper segment and within an area outlined by definite boundaries.

Before describing direct invagination of the descending mesocolon, attention is called to Figure 23 which shows all the divisions of the large bowel and their continuous mesenteries lying unfixed. Their progressive changes of position to ultimate placement in the abdominal cavity is indicated numerically. Figure 14 indicates that the descending mesocolon has its root at the midline of the dorsal wall of the embryo. Between the leaves of the peritoneal duplication which forms the descending mesocolon, the ascending branch of the left colic artery runs superiorly and somewhat laterally. The inferior mesenteric vein also lies in the mesocolon, and its main trunk is directed superiorly and mesially to combine with the superior mesenteric and splenic veins to form the portal vein. The artery and vein just mentioned usually run superiorly at some distance from the midline root of the mesocolon, thereby leaving a very considerable avascular space between the ascending branch of the inferior mesenteric artery and the inferior mesenteric vein and the root of the descending mesocolon. This interval is indicated by an area of stippling. The closer to the descending colon these vessels lie, the larger is the avascular interval of descending mesocolon.

It is our belief that in many cases of hernia into the descending mesocolon (so called left duodenal hernia), the upper jejunum and ileum in their migration to the left superior part of the abdominal cavity engage to a greater or lesser degree into the unsupported area of descending mesocolon just described. The mesocolon then falls to the left and fuses to the primitive posterior parietal peritoneum.

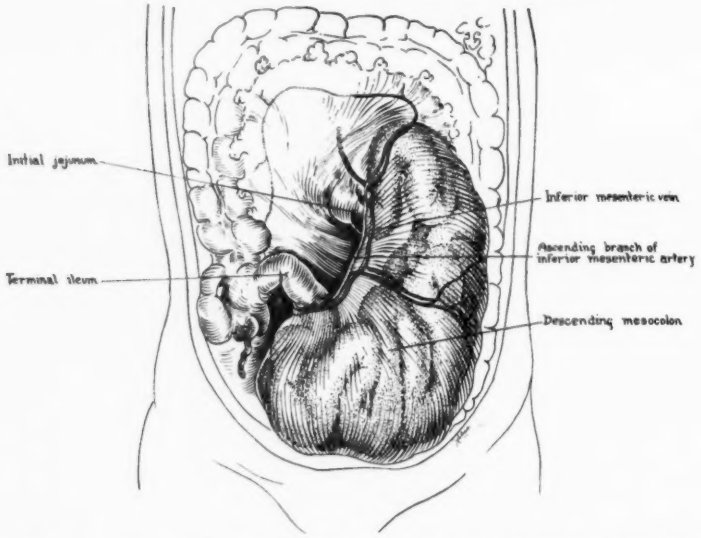

Fig. 26. Almost complete invagination of jejuno-ileum into descending mesocolon. This drawing represents the condition found in the case we are reporting. Compare it with Figure 1.

When the coils of small bowel invaginate the mesocolon, the vascular trunks located in the left margin of the avascular area resist invagination. The result is that these trunks then lie in the forward wall of the neck of a sac, all of which is derived from the avascular area of the descending mesocolon. Gradually all of the small bowel may enter the enlarging sac.

The series of changes resulting from the small bowel invagination of the descending mesocolon, while the mesocolon still is mobile, is illustrated in the series of cross sections in Figure 27. After all of the small bowel has entered the sac, the peritoneal surfaces outside the sac undergo fusion.

PLEA FOR A CHANGE IN NOMENCLATURE IN SO CALLED DUODENAL HERNIA

It must be evident that the duodenum, with the occasional exception of its terminal portion, is not an element in the hernial contents of the so called duodenal or paraduodenal hernia. Nor does the duodenum figure in any of the other varieties of so called duodenal hernia. It is the jejunum and ileum which are the contents of the sac. Inasmuch as part or all of the small bowel invaginates the descending mesocolon in the type of hernia under discussion, it is proposed that the so called left duodenal types of rupture be known rather as "hernia into the descending mesocolon."

In a succeeding paper, reasons will be adduced to justify a change in the name of that variety of intraperitoneal hernia known as "right duodenal hernia" to "hernia behind the ascending mesocolon." The anomalous rotation of the jejuno-ileum reported by Haymond and Dragstedt should not be classed with either of these varieties of intraperitoneal hernia; to that we wish to ascribe the name of "hernia into the ascending mesocolon."

SYMPTOMS AND SIGNS OF HERNIA INTO THE DESCENDING MESOCOLON

The hernia we have described caused no symptoms and produced no physical signs. Many herniæ with a similar lack of clinical manifestation have been found in a course of laparotomy for other conditions and during routine autopsy examination.

When, however, obstruction occurs in the bowel incarcerated within the hernial sac, an exact diagnosis of this type of internal hernia

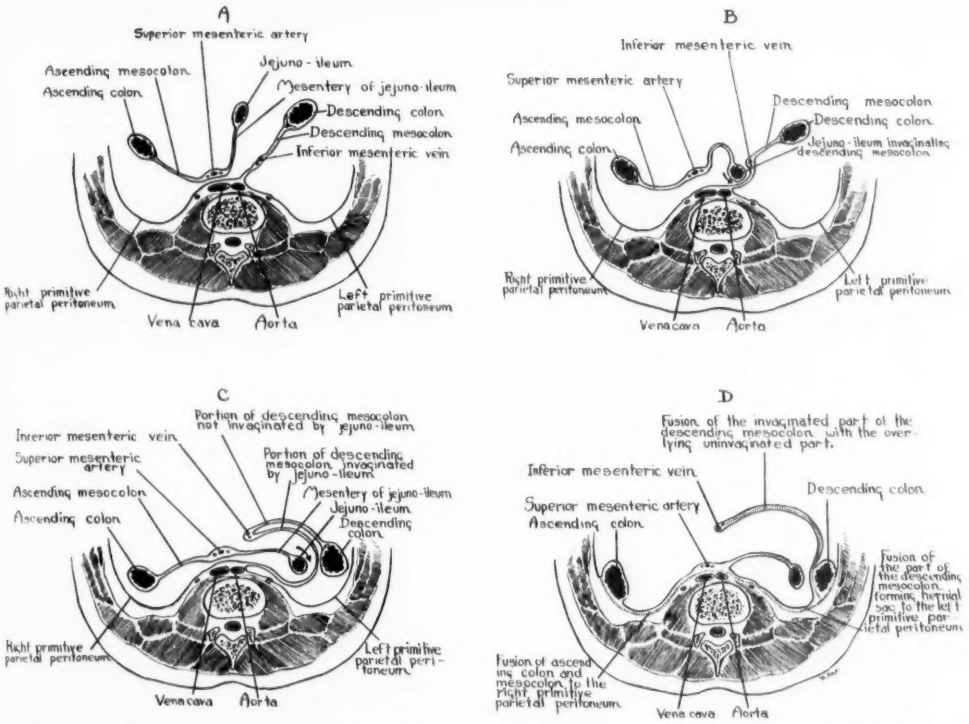

Fig. 27. Series of cross sections through region of development of hernia into descending mesocolon. A, In this section the jejuno-ileum and ascending colon are attached to a common mesentery and are mobile. The descending colon and mesocolon are mobile. Attention is drawn to the position of the inferior mesenteric vein in the descending colon. B, The jejuno-ileum has begun to invaginate the avascular area of the descending mesocolon between the inferior mesenteric vein and the midline attachment of the mesocolon, and a sac is in the process of formation. C, The jejuno-ileum has invaginated the avascular area of the descending mesocolon deeply and a larger sac is being formed. For clarity, only one loop of small bowel is shown in the sac. D, The opposed serous surfaces now fuse by the process of fetal agglutination. The colon and mesocolon now are fixed to the posterior abdominal wall.

sometimes has been, and in the future more often will be, made. Considerable diagnostic importance should be placed upon the history of chronic intestinal obstruction. One individual found to have this type of hernia suffered 40 different attacks of intestinal obstruction prior to the one which precipitated operation. If the signs and symptoms are only those of acute intestinal obstruction, an exact diagnosis is difficult to make.

Certain signs sometimes are present which are of great value. A soft tumor in the left side of the abdomen several times has been found in the reported cases of herniæ into the descending mesocolon, but the swelling usually has been misdiagnosed as an ovarian or a pancreatic cyst. The occurrence of one or more attacks of intestinal obstruction in addition to the presence of tumor should lead one to a consideration of the possibility of a intraperitoneal hernia.

At times the location and physical findings of the tumor are distinctive. Palpation may reveal the swelling as occupying the superior part or all of the left side of the abdomen. Percussion over the tumor should yield a resonant note, and the ausculatory findings may be those of gurgling peristalsis. The size and tension of the tumor mass and the degree of peristalsis depend upon the amount of obstruction in the incarcerated contents of the sac.

TREATMENT

The treatment of hernia into the descending mesocolon may be simple or extremely difficult. The operative results in the last 10 years have been much more favorable than those in earlier years—the consequence doubtless of more timely recognition of obstruction within the sac and earlier surgical intervention.

In several patients with small herniæ, the incarcerated bowel was drawn out without difficulty into the general abdominal cavity through the neck of the sac, which was closed by suture. In occasional instances, the serious condition of the patient has precluded the closing of the neck of the sac after withdrawal of its contents. On at least one occasion in a person so treated, adhesions developed spontaneously about the orifice of the sac, and the hernia did not recur. In some herniæ even of small size, manual reduction could not be accomplished; under these circumstances it might have been possible to loosen the neck of the sac by making a small incision in a superior and medial direction so that the vascular trunks in the forward walls of the neck could be avoided. It must be borne in mind constantly that the inferior mesenteric vein lies not only in the lateral margin of the neck of the sac but often courses medially in its superior portions in such a way as to preclude any incision in the neck of the sac.

In a very large hernia in which nearly all the jejuno-ileum lies within the hernial sac, the sac orifice usually is so constricted that the contents of the sac cannot be brought into the general abdominal cavity even when no adhesions at the neck of the sac are present. In the case we are describing, the neck of the sac was small and the adhesions about it were so adherent that painstaking, blunt, and sharp dissection were required to locate it. Had intestinal obstruction within the sac occurred, it would not have been possible, because of these adhesions, to have delivered the hernial contents into the general abdominal cavity. The only possible recourse in the treatment of such a situation would have been incision of the anterior wall of the sac (descending mesocolon) in an avascular area and the emptying of the dilated bowel by temporary enterostomy. After ascertaining and relieving the cause of the obstruction, the rent in the anterior wall of the sac should be closed.

SUMMARY

1. An instance is reported of herniation of practically all of the jejuno-ileum into the descending mesocolon (so called "left duodenal hernia").

2. The embryologic background of intestinal rotation and posterior peritoneal fixation is portrayed in order that the varieties of mechanism of production of this type of hernia can be understood.

3. A detailed description of the folds and fossæ about the terminal duodenum is given so that the rôle they play in the production of this hernia may be interpreted properly.

4. In this paper, it is suggested that hernia into the descending mesocolon occurs as a result of invagination of an area of the descending mesocolon by the jejuno-ileum while the descending mesocolon is mobile. A location in the descending mesocolon for the reception of the invaginating jejuno-ileum is indicated.

5. A change in nomenclature for the so called "left duodenal" type of intraperitoneal hernia is advocated. From the embryologic and anatomic viewpoints, this hernia should be known as a "hernia into the descending mesocolon."

6. The salient features in the recognition and treatment of hernia into the descending mesocolon are enumerated.

We are indebted to Dr. John B. de C. M. Saunders for his constructive criticism and suggestions in relation to the embryological discussion in this paper.

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THE USE OF VITAL STAINING AND WET FILMS IN THE DIAGNOSIS OF LESIONS OF THE CERVIX

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THE introduction by Schiller of a new and simple test for establishing the diagnosis of carcinoma of the cervix was of such interest and importance that the method has been tried at St. Thomas's Hospital upon a representative series of cervical lesions.

In the original paper Schiller pointed out that cancer of the cervix starts in the squamous epithelium in the vicinity of the os and spreads laterally and superficially from this point. The earliest changes consist of a proliferation of the squamous cells and the surface remains intact for some time before ulceration occurs. To locate these early patches of new-growth, a method of vital staining which differentiates normal from malignant epithelial cells was devised, and small portions of suspicious material were removed for microscopy. As regards this method of vital staining it was pointed out that the normal squamous cells of the cervix contain granules of glycogen, and that these granules could be stained vitally with Lugol's iodine. Schiller also stated that in some pathological conditions, and notably in early cases of carcinoma when the surface had not ulcerated, glycogen was absent from the squamous cells and vital staining did not occur. He claimed, therefore, that by using Lugol's solution, it is possible to make a diagnosis of carcinoma earlier than by the usual clinical means.

Since the publication of this work in SURGERY, GYNECOLOGY AND OBSTETRICS, several other investigators, particularly Graves and Frank, have endorsed these views and have stated that they are very much impressed by the value and possibilities of the method. Our own opinions are not in entire agreement with those which have been recorded by others, and the object of this paper is to state the value of vital staining as applied to lesions of the cervix in general; the drawbacks and difficulties in cases of carcinoma; and to advocate the use of "wet films" for the immediate investigation of pieces of tissue removed for biopsy.

The cases we have examined have practically all attended the Out-Patient Department, and include normal cervices, erosions, ectropion of the canal, extrusion of the mucosa, polypi, traumatic conditions, and carcinomata both of the cervical canal and vaginal cervix.

The technique we have adopted is as follows: A speculum is passed after bimanual examination has been done, and the cervix is examined macroscopically. Mucus or other secretion is carefully wiped away and the surface of the cervix and vaginal vault is painted with Lugol's solution. The excess of iodine is sponged away and the reaction of the parts noted. In every instance small pieces of tissue from suspicious areas are removed and from these wet films are made immediately. The preparation of these films will be discussed subsequently.

The material for biopsy was obtained by a special punch[1] which we recommend to any who may wish to secure such material easily without causing inconvenience to the patient. Small pieces can be removed from the cervix without pain, even in cases of cervicitis, and on only one occasion have we had any trouble from bleeding.

Clinically our cases fall into three main groups when judged by the reaction of the cells to the vital stain.

In the first group are those in which the cervix and vaginal vault stain diffusely, evenly, and deeply. The reaction occurs almost as soon as the solution has been applied. This is the reaction of the normal cervix and apart from certain qualifications is fairly constant.

[1] Made by Messrs. Allen and Hanbury to our order.

The depth of the stain varies to some extent with the age of the patient, and, in the light of the work of Cruickshank and Sharman, this is not surprising, for they have demonstrated that the glycogen content of the epithelium steadily diminishes after the menopause, until in extreme old age, it is very difficult to find any glycogen in the cells at all. This fact must be important in interpreting results of vital staining in old people. Again there are many cervices which show clinical evidence of tears and cervicitis which stain in the normal way, and finally it must be remembered that since the columnar epithelium of the cervical canal does not contain glycogen there will be a ring of unstained tissue round the os in cases suffering from ectropion of the mucosa.

The second, and perhaps the most common group of cases, is those in which the cervix presents a mottled appearance, due to patches of light brown or lemon yellow color among the normal dark stain. A peculiar feature of these patches is that they do not correspond to clinical lesions which are apparent macroscopically; on several occasions we have noted these patches in what we had judged to be a normal cervix. One is therefore forced to the conclusion that other factors influence vital staining apart from pathological conditions. The mottled cervix is most often seen in cases of erosion, polypoid conditions, cervicitis, and in carcinomata which have ulcerated. It has been pointed out by other observers that pus on the surface of a lesion stains brown or black with the iodine, and it is important not to overlook cases of early malignant ulceration when obscured in this manner. In our series we have seen only one case of carcinoma in which the surface of the growth had not ulcerated, and this was a patient who was pregnant and who was found to have an early polypoid growth involving the posterior lip of the cervix. Lugol's iodine gave a mottled appearance, and we were in doubt as to the correct diagnosis until the wet film had been examined. Sections made from the tissue removed confirmed the diagnosis and the tumor proved to be a squamous cell carcinoma. This case supports the view which has been expressed by others that is that pregnancy may interfere with the test.

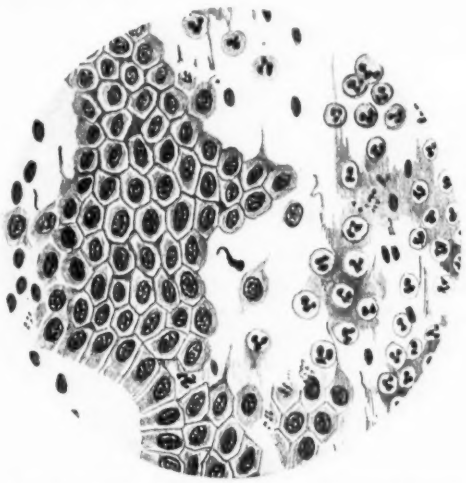

Fig. 1. Film showing plaque of normal cervical mucosa and acute polymorphonuclear inflammation. The regularity of the nuclei, the presence of cell membranes between the individual cells, and the even staining are the features which indicate the benign nature of this epithelium. ×330.

The third clinical group is those in which there are absolutely white and unstained areas, and we consider that the distinction between this complete absence of staining and the lightly stained areas in the mottled cervices described has not been sufficiently emphasized. In these areas the cells are not even colored light brown by the iodine, and they stand out in marked contrast to the dark brown of the vital stain. These areas according to Schiller indicate—

1. The presence of carcinomatous layers or incipient cancer.

2. The presence of hyperkeratosis, a result of prolapse.

3. The presence of hyperkeratosis of luetic origin.

4. Desquamation of the superficial layers of the epithelium by trauma.

In our series we have found that the lesions mentioned in this list are more frequently associated with a mottled or light staining, and those in which we have found white patches have been fungating carcinomata, areas of trauma which extended deeper than the epithelial layer, and occasionally in old scars and fibrous lesions.

Fig. 2. Normal squamous epithelium showing the gradual change from the basal layers to the superficial desquamating cells on the surface. ×75.

Considering these results as a whole they show that the staining properties of Lugol's iodine are inconstant and difficult to interpret and are influenced by factors apart from pathological lesions. When it is remembered that cases of cancer of the cervix in the preulcerative state are rare and that normal staining of the cervix does not exclude a carcinoma low down in the cervical canal, it becomes obvious that vital staining is not as valuable a method of investigation as appears at first sight. Indeed, we have come to the conclusion, after careful trial of the method, that visual and manual examination, supplemented in difficult or doubtful cases by a wet film is the most likely to give an accurate picture of the various conditions we have treated.

Wet films are of use in this type of work because it is possible to give a diagnosis in the Out-Patient Department while the patient waits, 10 minutes is all that is required in an urgent case, and because experience of this technique which has been in use at St. Thomas's Hospital during the last 8 years has convinced us that in the hands of those who have studied the question, the interpretation of films is accurate and reliable, their preparation is simple, and the materials required are available in any pathological laboratory. The reader is referred to papers (2, 3, 7) which have been published on this subject, for they will serve as a guide to the technique of preparing wet films, and the descriptions of preparations made from pathological conditions of all kinds elsewhere in the body give an idea of the value and limitations of the method. We have made wet films from every case in our series and a definite diagnosis has always been possible even in those instances where the staining of the cervix by Lugol's solution has been difficult to interpret or even deceptive.

The tissue removed with the punch must be teased out upon the slide before it dries, for the appearance of the cells alters rapidly if the tissue be kept for any length of time, and it is essential that saline, preservatives, or other fluids are not used, as diagnosis is not possible if autolysis has occurred. Sometimes it is difficult to make a good film from these cases because the piece of tissue available is small and may be very fibrous; the best results are obtained by fixing it on the slide with a mounted needle and then teasing it with a scalpel which should have a broad blade. If the method is used in conjunction with Schiller's technique, as we have done, it will be found that the epithelium which has taken the vital stain is decolorized at once by the Schaudinn's solution, and consequently the iodine does not affect the subsequent staining with hæmalum and eosin.

In order to interpret wet films made from the cervix it is necessary to have a knowledge of the normal histology of the part and the changes to which the cells are liable in health and disease.

In the normal cervix the junction of the squamous and the columnar epithelia is sharply defined and lies at the margin of the external os. The columnar cells vary in number and appearance not only with the age of the patient but also according to the stage of the menstrual cycle. It is usual to find them distended with mucus in vacuoles; this can be confirmed by staining with muci-carmine. On the surface the epithelium is more regular and less distorted with globules of secretion than in the deep glands, and in old age the number of cells is diminished and the glands may be

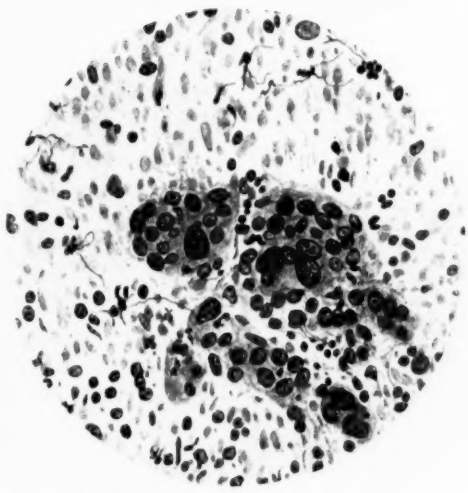

Fig. 3. Carcinoma of cervix. The plaque of epithelial cells in this film shows irregularity in the size and shape of the nuclei, an absence of cell membranes between the individual cells, and a tendency of the nuclei to separate from each other. Compare this film with Figure 1. ×280.

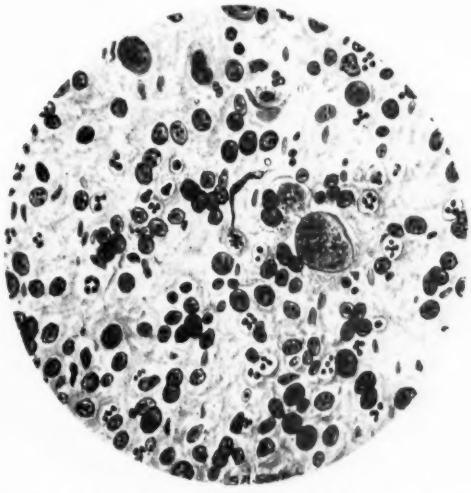

Fig. 4. Carcinoma of cervix. The malignant cells are completely separated from each other. The nuclei vary enormously in size and shape. There are some mitotic figures and there is evidence of diffuse polymorphonuclear inflammation. ×280.

absent. The squamous epithelium of the adult is similar to that elsewhere in the body consisting of a basal layer, a zone of prickle cells, and a mass of desquamating cells on the surface. In films the granules of glycogen cannot be seen, but in sections fixed in absolute alcohol and stained by Best's technique they are found easily and are scarlet. They are concentrated for the most part in the deeper layers of the epithelium.

The height and depth of the squamous epithelium vary in health and are not necessarily connected with pathological conditions, they bear a close relationship to the glycogen the cells contain, and this is dependent upon ovarian activity.

The epithelial cells rest upon a stroma composed of spindle cells and small blood vessels.

In films the various epithelial elements and the stroma are seen and the differences which occur in the normal cervix must not be forgotten.

The columnar cells in the film are found either in plaques resembling a tessellated pavement, or as a typical row of epithelial cells with an even, round nucleus at the base of each cell. The mucus in these cells causes variation in their size and shape, but the regularity of the nuclei and the definite cell membrane are the features upon which the diagnosis of benign epithelium is made. Separation of the cells tends to occur if there is much mucus.

The squamous epithelium nearly always separates into two parts: the superficial desquamating cells break away in plaques from the prickle cells and basal layer and these plaques stain pink, whereas the deeper cells take up hæmalum strongly and are blue. In the presence of acute inflammation it is not unusual for the squamous and columnar cells to be phagocytic and we have several films showing these cells engulfing polymorphonuclears. If this occurs the cells are much larger than normal, but the enlargement is confined to the cytoplasm and the nuclei remain small and regular.

The stroma is represented by groups of fibroblasts and by isolated spindle cells. In cases of cervicitis there are areas of granulation tissue and polymorphonuclear leucocytes are plentiful. It is occasionally possible to demonstrate capillaries in films made from granulation tissue.

The main application of this method is to distinguish between inflammatory and neoplastic conditions. We have noticed that almost every cervix we have examined has shown a marked degree of polymorphonuclear infiltration. This inflammation does not necessarily produce pain or symptoms, but is probably a cause of the excess of mucus secretion in chronic cervicitis. We have found inflammation not only in cases in which surface ulceration is present, but in patients with an intact epithelium covering the cervix.

A diagnosis of carcinoma is made upon the appearance of the malignant cells themselves and does not depend upon infiltration of the neighboring tissues or the presence of metastases. The points upon which this diagnosis is made have been described fully elsewhere and the reader is referred particularly to the most recent paper on the subject in the *British Journal of Surgery* (2).

In recommending wet films as an aid to the diagnosis of lesions of the cervix, we desire to thank Professor Dudgeon who, with C. V. Patrick, introduced this method in 1927 and has helped us by examining the majority of the films we have made.

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STUDIES ON PERIPHERAL VASCULAR PHENOMENA

IV. Finger Volume Changes in a Patient Showing Raynaud's Phenomena

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EVER since Raynaud (5) described the clinical entity carrying his name, this disease has been the subject of a great deal of study.

We wish to present a case upon which special studies were made that may throw more light upon the basis of the peripheral vascular phenomena which occur in this obscure disease.

REPORT OF CASE

Mrs. I., 37 years old, consulted one of us (Hedges) in November, 1933, for stiffness of the fingers. Her chief complaints were inability to play the piano and some pain and stiffness of the toes, when she walked. Mrs. I. states she had iritis of right eye in 1929 and following this her right ankle and wrist became swollen and tender. A diagnosis of arthritis was made and she had some teeth removed and a tonsillectomy performed. The arthritis subsided but she soon began to experience some stiffness of the fingers of both hands. No attention was paid to this condition until about April, 1933, when Mrs. I. was forced to stop playing the piano.

Previous to the above history patient had enjoyed good health. Her husband died suddenly in 1933 of septicæmia. The family history was negative. There was no history of malaria. Physical examination is briefly as follows: height 5 feet, 6 inches; weight, 147 pounds; blood pressure—right arm 110/68, left arm 94/68; pulse, 88.

Color of both hands was poor—dusky color. The skin was tense over the fingers, especially of the right hand. The first and second fingers of right hand were stubby but did not show a true clubbing. The hands were cold. She was unable to flex the fingers of either hand, especially the right hand. There was no muscular atrophy of the arms. The hands appeared small and perspired freely in palms. Some slight nutritional disturbance of finger nails was present. Pain was present across the knuckles when hand was pressed. There was a thickening of the skin of the ventral surface of the hands. There was a definite line of demarcation just distal to the middle joint of the fingers of both hands on immersion in cold water. This pallor remained for about 2 minutes and was replaced by cyanosis (Fig. 1). There was no disassociation of heat and cold sense on either arm or hand.

The lower extremities were practically normal although there was some slight enlargement of the joints present. The blood was normal, with the exception of a slight anæmia—3,000,000 red blood cells. The urine was negative, no Bence Jones protein present. Blood chemistry analysis, normal; calcium, 9 milligrams per cent; phosphorus, 2.8 milligrams per cent. The blood viscosity was normal. The electrocardiogram was normal.

Roentgenograms were made of both hands (Fig. 2).

One showed considerable loss of distal phalanges, especially of the right hand.

Roentgenograms of the feet showed no such destruction of bone. There was no cervical rib present and X-ray examination of the long bones, head, and pelvis, showed no systemic bone involvement.

Studies on the peripheral circulation were made with a special device previously described by Johnson. This device measures the finger volume change associated with the heart beat. Skin temperature changes were made with the ordinary thermocouple, and color changes were observed by comparison with the color charts as devised by Lewis.

The finger volume changes at room temperature were uniformly lower than usually observed in the normal human being. Also the skin temperature was uniformly lower than observed in the normal (Figs. 3, 4, 5).

Immersion of the hands in hot water (45 degrees C. for 10 minutes) caused a moderate increase in the finger volume changes (Fig. 3).

Immersion of the hands in cold water (10 degrees C.) caused a reactive hyperæmia associated with increased finger volume changes without cyanosis.

Occasionally it was possible to induce a spasm of the peripheral vessels by immersion of the hand in moderately cold water (between 13 degrees and 18 degrees C.). Associated with this, the fingers assumed the color changes characteristic of Raynaud's phenomenon. This is in keeping with the observations of Lewis, that the optimum temperatures for inducing a spasm lie between 13 and 18 degrees C.

The color changes of Raynaud's phenomenon were most easily induced by having the patient go out of doors and expose her hands to moderately cold air and Figure 4 shows graphically the changes which occur during the spasm. It is shown by these charts that even in the very cyanotic stage the finger volume changes were greater than the control and subsided to a very low level in about an hour. As will be seen from the charts the skin temperature did not bear uniform relationship to the color changes or the finger volume changes in this particular case.

Fig. 1. Photograph of the patient's hands during an attack. Note the cyanosis of the fingers of the right hand up to the first interphalangeal joints. The ends of the fingers are stubby but a true clubbing is not present. This is particularly true in the right hand where the absorption of the terminal phalanges is most marked (see Fig. 2).

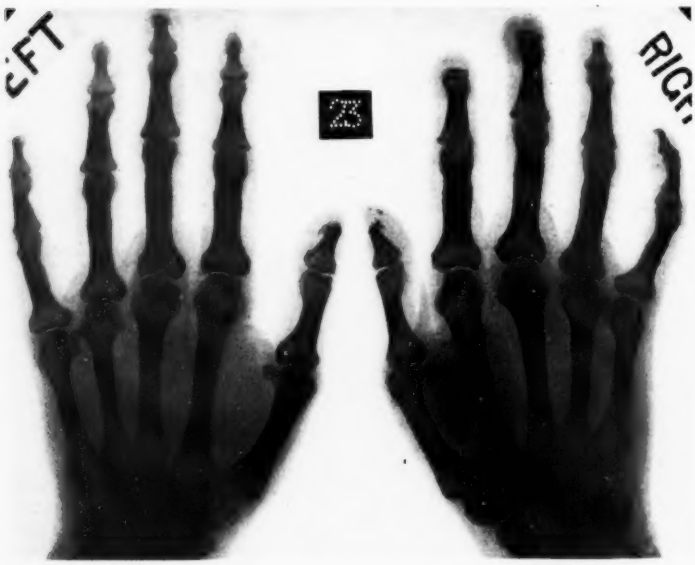

Fig. 2. Roentgenograms of the bones of both hands showing the absorption of some of the terminal phalanges. This is most marked on the right.

Treatment. This patient was given tissue extract because it was supposed to cause vasodilatation, but it has been shown by Roth and Barber not to exert its effect by vasodilatation. These authors think that its action is directly on the muscles supplying a deficiency due to inadequate blood flow.

The result was not very satisfactory in this case, although, if a spasm was induced by exposing the hands to cold air, tissue extract would cause a color change, i.e., from cyanosis to a more normal color.

After several months' treatment with tissue extract, Mrs. I. noticed improvement in her ability to move the hands. No noticeable improvement was noted in the scleroderma. The vascular spasms were of about the same intensity at the termination of treatment and about the same duration. The tissue extract would cause a color change lasting about 2 hours. Figure 5 shows the results upon the skin temperature, color, and peripheral pulse volume. There was improvement in the colors of the fingers of the right hand but no increase in the pulse volume changes. From this result it would be difficult to

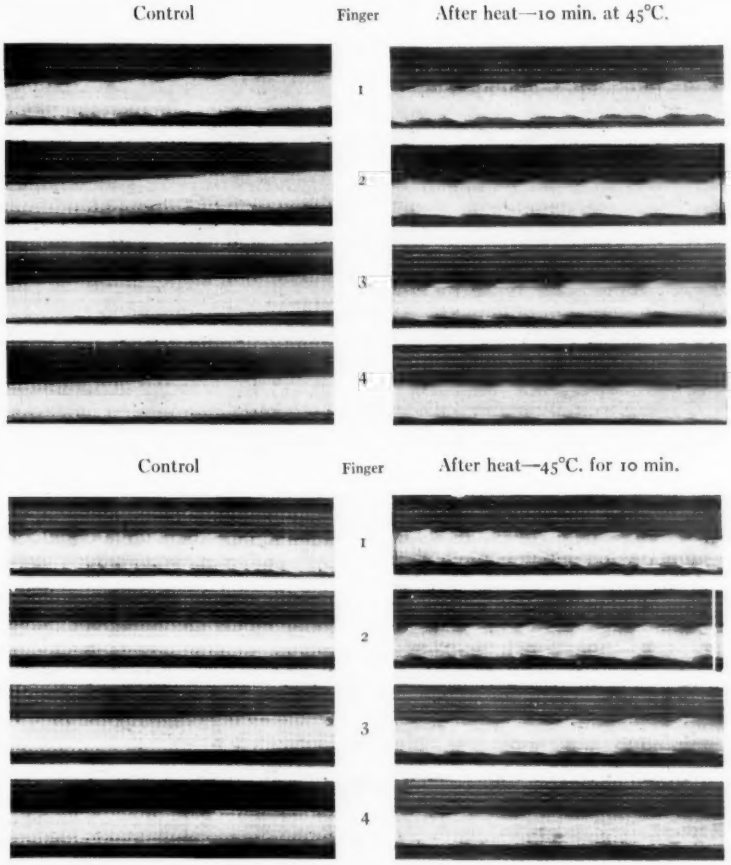

Fig. 3. The finger volume changes of the fingers before and after the application of local heat. (Immersion of the hand in water at 45 degrees C. for 10 minutes.) The upper record shows records from the fingers of the right hand and the lower are the left. Note the very low amplitude in the control records indicating that under normal environmental conditions the circulation is less than normal. The local heat relaxes the peripheral vessels and allows for an improved circulation.

draw any conclusions regarding the value of this medication.[1]

The classification of this case as one of Raynaud's disease is open to some question, because as Lewis states: "Thus, it is quite possible that patients who in the first instance display attacks of cyanotic fingers and subsequently acquire a general scleroderma are essentially of the same type; but as we have yet no proof that the vessels in these cases respond directly and unusually to temperature, and as we do not understand how the general scleroderma occurs, it is quite clearly desirable that this class of case should be kept apart until it has been submitted to suitable investigation" (4). On the other hand, we think this case would fall into group 2 of the 204 cases of Raynaud's disease as reported by Allen and Brown (1). Kolodny (3) reported a case with symptoms and X-ray findings similar to the one we have described who received con-

[1] Later experiments have shown that it is possible to demonstrate a vasodilatation, as evidenced by increased finger volume changes, by using larger doses of tissue extract and waiting a longer period. The maximum change occurs in about ½ hour and the effect wears off in about 1 to 2 hours, depending upon the dose.

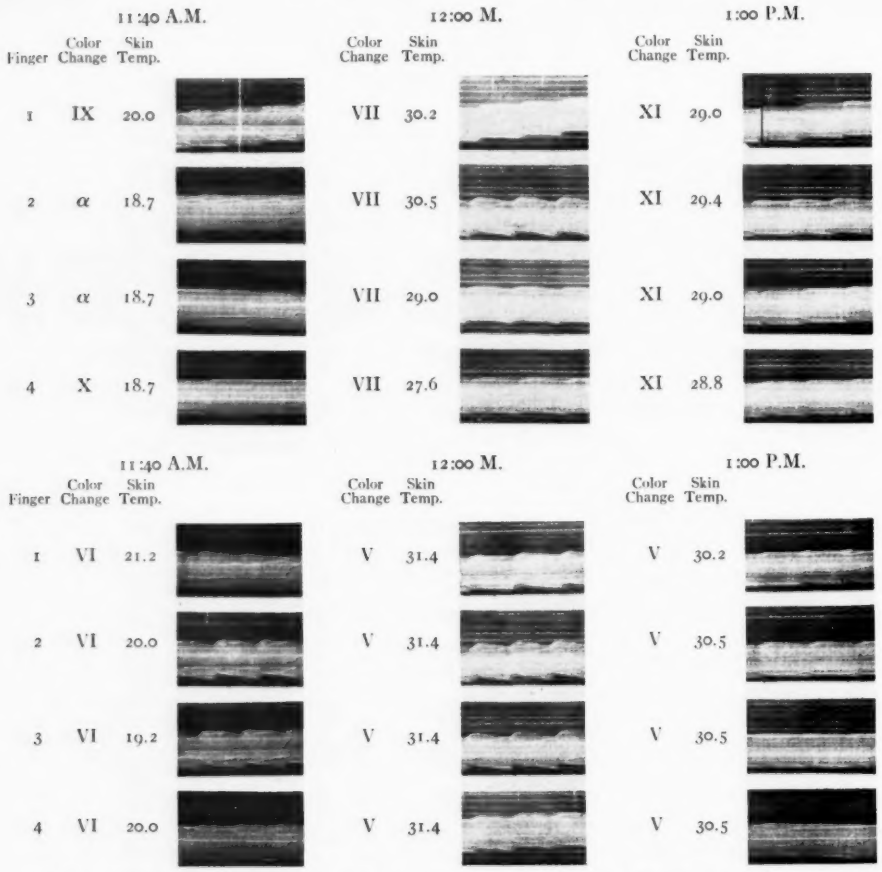

Fig. 4. The finger volume changes in the fingers of both hands just following vascular spasm. The upper record is the right side and the lower the left. The skin temperatures are shown and the Roman numerals and letters represent the colors of the fingers as compared with the color charts devised by Lewis. Note that in the cyanotic stage the fingers become warm and the pulse volume changes are increased indicating an increased circulation. This subsides in about an hour to a very low level, but the skin temperature changes lag considerably behind this change.

siderable relief from a cervical ganglionectomy and ramisectomy. We consider our case one of Raynaud's disease complicated with scleroderma and sclerodactylia.

The symptoms in our patient are quite characteristic of Raynaud's disease:

There is a long history of rather painless attacks of cyanosis of the fingers up to the first interphalangeal joints (Fig. 1). The disease is symmetrical, i.e., it involves the fingers of both hands and also less severely the toes of both feet. The attacks are initiated most easily by moderate cold such as temperatures between 13 and 18 degrees C. An attack is more easily initiated by exposure to cool air than by immersion of the hand in cold water. As was the case with most of Lewis's patients, the phenomena of pallor did not occur, but the fingers became cyanotic or violet and as recovery took place became red and finally assumed their usual color.

The plethysmographic studies lend support to the diagnosis of Raynaud's disease. The pulse volume tracings in the absence of an

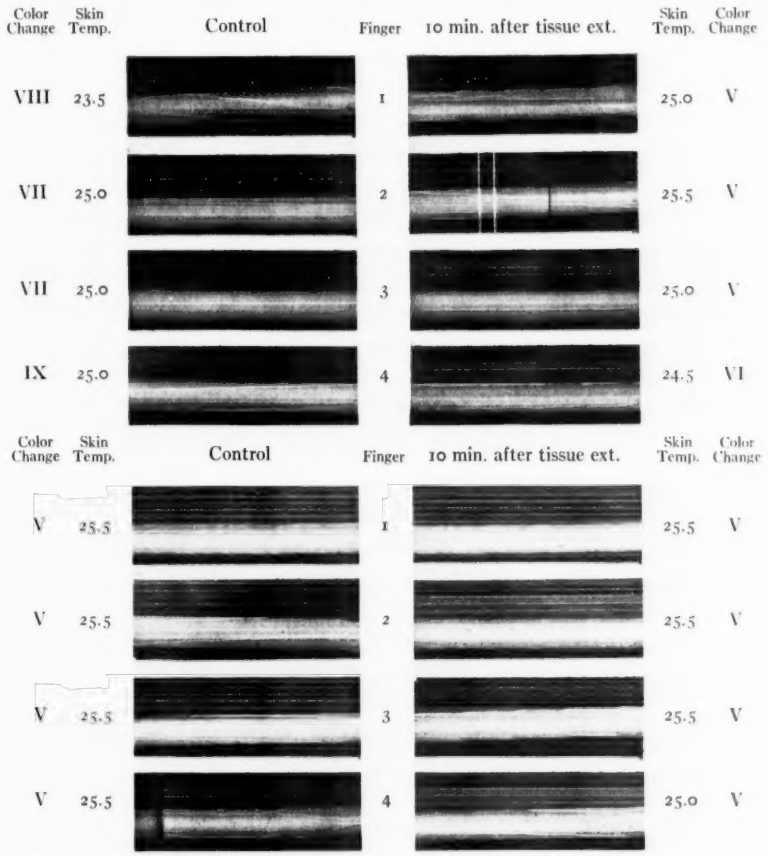

Fig. 5. The effect of tissue extract on the peripheral circulation, temperature and color changes. The skin temperatures did not change but there was an improvement in the color of the right hand, above. There was no improved circulation following this medication.

attack were uniformly lower than in normal individuals indicating a large vasospastic element (Figs. 3, 4, 5). This could be relieved by immersion of the hand in hot water (Fig. 3). When an attack occurred and the blue color appeared, the skin temperature rose rapidly and the pulse volume changes increased. As the attack subsided the pulse volume changes decreased to a subnormal level but the skin temperature did not parallel these.

Operation with a cervical ganglionectomy and ramisectomy was advised, but the patient refused. It was felt that the interference with the heat regulating mechanism of the upper extremities as evidenced by the increased skin temperatures following this operation would render her less liable to attacks.

SUMMARY AND CONCLUSIONS

We have presented a case of Raynaud's disease complicated with scleroderma and sclerodactylia.

Circulatory studies were made with a special finger plethysmograph which indicate that in this case the peripheral circulatory changes were as follows:

1. The circulation was persistently less than normal in the fingers even in the absence of an attack.

2. A peripheral vasodilatation could be induced with local heat.

3. During the cyanotic stage of Raynaud's phenomenon the circulation was increased.

4. Tissue extract improved the color of the fingers, but did not induce vasodilatation.

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ELECTROSURGICAL ASEPTIC INTESTINAL ANASTOMOSIS[1]

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ELECTROSURGICAL methods as applied to the hollow viscera are still in the experimental and very early practical clinical stages and further studies are imperative before their actual value can be established. With this in mind we have performed several different types of aseptic intestinal anastomosis experimentally and have applied the procedure clinically in man.

This paper is a presentation of the experimental technique used, the surgical pathological physiology of the procedures, and the suggestions of their clinical application in man.

Ward (2) at Kelly's suggestion performed anastomosis by electrocoagulation as follows:

> The loops to be joined were grasped with the usual anastomosis clamps drawn together parallel. Two layers of Lambert suture were used to approximate the posterior walls of the proposed stoma. In lieu of making an opening into the lumina of the loops, the intended sites of opening were coagulated by inserting a needle electrode carrying a biterminal current down to but not through the mucosa, relying on the coagulation at the tip of the needle to destroy the mucosa also. The needle not piercing the mucosa, obviated any immediate contamination from the bowel lumen. The two anterior layers of sutures were then laid, completing the operation. In from 24 to 48 hours, the coagulated tissue sloughed through and completed the anastomosis. The technique was fairly satisfactory in the intestinal anastomosis but was not to be relied upon in the stomach with its redundant mucosa which was coagulated ineffectually. We have not yet tried this procedure on man and feel that it is not applicable where there is an immediate need of an anastomosis.

Briggs (J.E.) and Whitaker (L.R.) later reported similar experiments performed independently of Ward's. Their technique calls for only one row of sutures to avoid leakage, as the delayed opening allows thorough sealing of the edges of the anastomosis by the fibrinous exudate before there is any chance for contamination.

In an effort to perfect an aseptic anastomosis technique that would immediately open the loops at operation, W. W. Walker devised a small hollow wooden instrument (Fig. 158) about the diameter of a lead pencil and 2 inches long. A groove was cut on each side, through which two pointed prongs could move from one end to the other by means of a stiff wire extending out of the end of the instrument. At operation, the posterior sutures were laid first, the instrument being placed between the loops so that the sharp prongs would each pierce the wall of one loop. The anterior sutures then closed the loops over the instrument. As a biterminal current was applied to the wire projecting from the end of the instrument, traction was made, drawing the prongs through the walls and cutting the tissues as they passed. On removing the instrument, the incisions were found to be open and dry, all vessels having been coagulated. The impractical feature of this instrument lies in the inability to remove it from between the loops of bowel in a completely aseptic fashion: as at present constructed, it must be withdrawn from its position into the sterile field. Attempts have been made to obtain an insulating substance out of which a collapsible instrument could be constructed, which would fall within the lumen of the intestines and later be passed. The material out of which such an instrument is made must of necessity be sterilizable and capable of being worked up into a tiny device. These obstacles are technical and are being studied.

The following operations have been successfully performed by us experimentally: (1) gastro-enterostomy, (2) cholecystgastrostomy, (3) anastomosis of small intestine to small intestine, (4) anastomosis of small intestine to large intestine, (5) anastomosis of large intestine to large intestine, (6) anastomosis of ureters to large intestine. On man we have

[1] From the Service of the Third Surgical Division, Bellevue Hospital, Dr. Arthur M. Wright, Director, and Department of Experimental Surgery, New York University and Bellevue Hospital Medical College

successfully performed a side-to-side anastomosis in the large bowel.

APPARATUS AND INSTRUMENTS

Any inexpensive apparatus carrying coagulating current which can be set to 1000 milliamperes and ranging to 1500 milliamperes can be used. We have employed an ordinary commercial model carrying the above range of coagulating current. To set the apparatus the milliameter is connected to the diathermy pole and the spark gap advanced until the milliameter registers 1000 milliamperes. The degree of spark gap advancement necessary to raise the coagulating current to 1500 milliamperes is then noted. The diathermy connection is transferred to the coagulation pole and the other connected to the indifferent electrode. Since 1000 milliamperes is the amount of current of choice, the spark gap is reduced to that point in the apparatus.

This amount of current gives a slow, even, and penetrating coagulation without causing distant injury and avoids loss of control. The coagulated tissue first turns white "parboiled," about the same color as the coagulated albumin of a hard boiled egg. If coagulation is continued further the "parboiled" area turns a light grey, then a dark grey, followed by charring. In ordinary work on the small and large bowel the light grey color is the optimum stage of coagulation. In doing work on thicker viscera as the pylorus and stomach, a little darker grey is the optimum stage of coagulation. Therefore one can regulate quantitatively by increasing the milliamperage, the speed, and degree of coagulation.

The electrodes are the unipolar (Fig. 1a), the bipolar (Fig. 1b) and the multipolar (Fig. 1c). The unipolar electrode consists of an ordinary non-cutting intestinal needle affixed to a holder, and used in conjunction with an indifferent electrode which is placed under the patient to complete the circuit. While this electrode can be used for any type of work, it is slow and tedious when long lines of coagulation are to be made. Therefore, it is reserved for fine point coagulation as in performing operations on the gall bladder and ureters.

The bipolar electrode (Fig. 1b) is the electrode used for electrocoagulation of tonsils and sold commercially for that purpose. The points of the electrode are introduced through the serosa and embedded in the muscularis of the portion of the intestine to be coagulated and gives an area of coagulation 5 millimeters in circumference and through all the layers of the bowel. In doing work on the stomach or pylorus, the needle points must be introduced deep into the muscularis to insure coagulation of the mucosa. One can readily see that by point-to-point coagulation linear, circular, etc., coagulation can be fashioned.

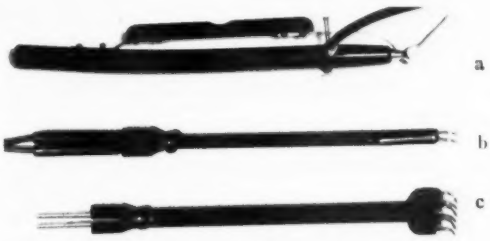

Fig. 1. Electrodes. a, Unipolar; b, bipolar; c, multipolar.

The multipolar electrode (Fig. 1c) was devised by one of us and constructed at the university machine shop. It is in reality a six pronged bipolar electrode giving a line of coagulation 2.5 centimeters in length and through all the layers of the bowel. The same technique is employed as in the use of the bipolar electrode. Long lines of coagulation can be performed very quickly, thus greatly reducing the time of the procedures.

TECHNIQUE

The general principle of the operation for aseptic intestinal anastomosis is illustrated in Figure 2. The loops to be anastomosed are approximated by a seroserous continuous suture of 20 day chromic No. 00 atraumatic, as is done in the first step of any conventional type of anastomosis (Fig. 2a). With the bipolar or multipolar electrode two lines of coagulation are performed on what normally would be the lines of cautery incision in the conventional technique. The multipolar electrode is the instrument of choice attached to 1000 milliamperes coagulation current. The prongs are introduced through the serosa and into the muscularis. The coagulating points

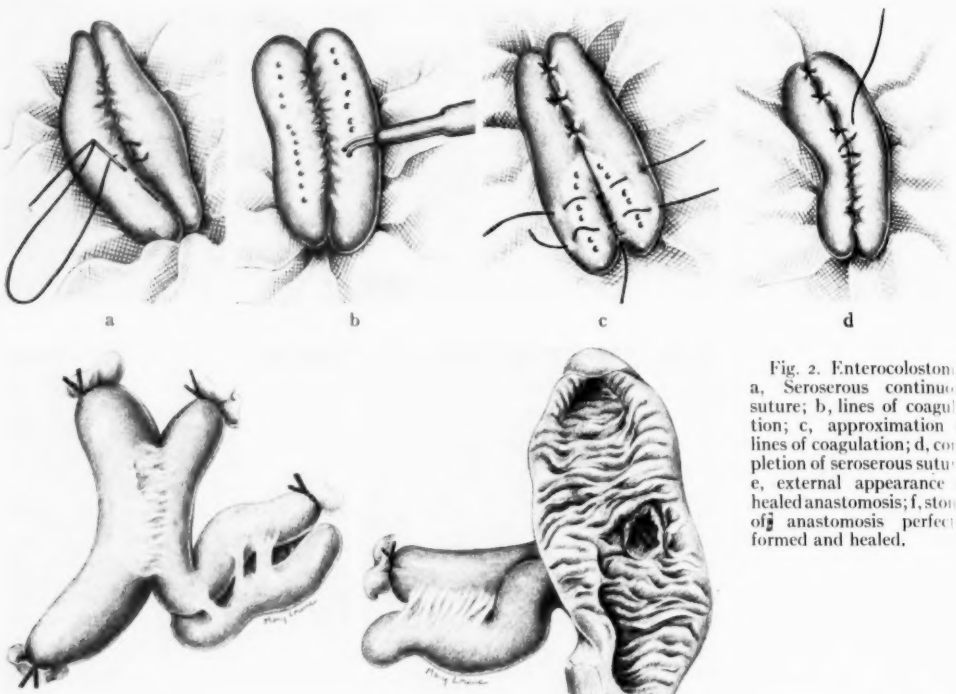

Fig. 2. Enterocolostomy. a, Seroserous continuous suture; b, lines of coagulation; c, approximation of lines of coagulation; d, completion of seroserous suture; e, external appearance of healed anastomosis; f, stoma of anastomosis perfectly formed and healed.

must enter 1 centimeter away from the seroserous suture line and must begin and end 1½ centimeters proximal to the ends of the seroserous line of suture (Fig. 2b). The circuit is completed by means of a foot switch controlled by the operator. The area around the electrode gradually turns white "parboiled" and becomes as hard as shoe leather and then turns light grey. When the white "parboiled" color appears, the area is carefully watched as it quickly turns to a light grey which represents the optimum point of coagulation. The foot switch is then released and the electrode advanced further to a new segment of the coagulation line until the two lines of coagulation are completed as shown in Figure 2b.

The two lines of coagulation are approximated by a series of two or more Lembert sutures of plain catgut as shown in Figure 2c placed 1½ centimeters apart. The seroserous suture begun in step 1 (Fig. 2a) is now completed to bury the two apposed lines of coagulation (Fig. 2d).

In the first 24 hours, there is an inflammatory reaction which completely seals the area between the lines of coagulation and the seroserous suture. When the slough begins to separate at the end of the 24 hour period no spilling can take place into the peritoneal cavity. Gas and liquid usually pass through in 36 hours and within 48 hours the anastomosis allows also solids to pass. In 7 days the anastomosis is completely formed and healed.

The animal made an uneventful recovery and the specimen obtained showed the complete external healing (Fig. 2e) and perfectly formed and healed anastomosis (Fig. 2f). It is of interest to note that this represents a successful anastomosis between the third portion of the duodenum and the ascending colon. These two loops were chosen because the anastomosis of these two loops represents the

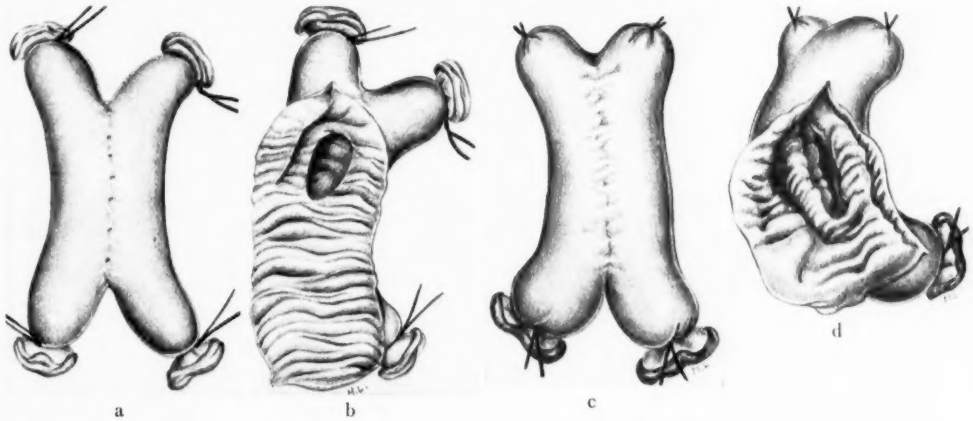

Fig. 3. Entero-enterostomy. a, External appearance of healed anastomosis; b, healed and functioning stoma; c, healed external appearance of entero-enterostomy following intestinal resection; d, stoma healed and functioning.

severest test that can be given any technique for anastomosis. Three controls in which the conventional suture methods were employed and the most scrupulous asepsis and technique were used, all promptly died with infection and secondary leakage. One must remember the strong digestive power of the duodenal contents and the virulence of the fæcal material to understand readily the poor results that are obtained by the use of the conventional method.

The final length of the completed anastomosis is usually two-thirds the length of the original line of coagulation. If a 2 inch anastomosis is desired as a final result, the line of coagulation must be 3 inches in length. No special aseptic precautions are necessary; there is no spilling, no hæmorrhage, and the time for the operation is one-third the amount used in doing the conventional type of operation.

Anastomosis of jejunum to ileum 2 weeks after operation is shown in Figure 3. This technique is most adaptable and successful in this type of anastomosis. Figure 3a demonstrates the perfectly healed seroserous surfaces and Figure 3b, the perfectly formed, healed, and functioning anastomosis. As in all our experiments, no pre-operative or postoperative special precautions as to diet, etc., were taken in order to give the technique a severe test.

INTESTINAL RESECTION AND SIDE-TO-SIDE ANASTOMOSIS

Twelve inches of ileum were resected and the ends of the resected bowel were inverted in the usual way. A side-to-side anastomosis was performed as shown in Figure 2. The dog never refused food and never had any symp-

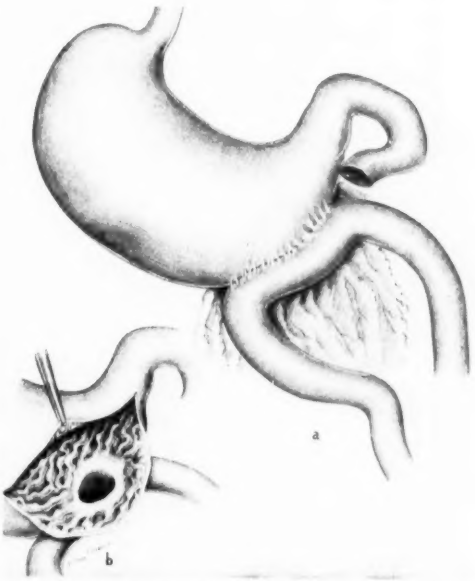

Fig. 4. Gastro-enterostomy. a, Healed external appearance of anastomosis; b, healed and functioning stoma.

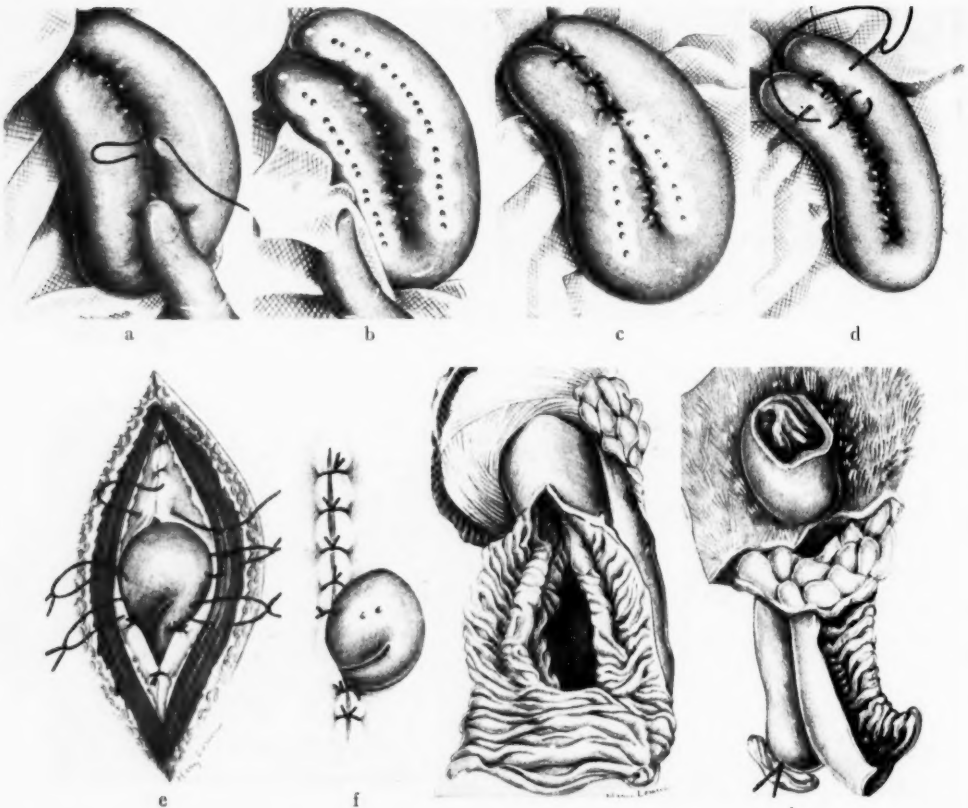

Fig. 5. a, Seroserous suture line; b, lines of coagulation; c, apposing the lines of coagulation; d, completion of seroserous line of suture; e, closure of abdomen and fixation of colon to peritoneum; f, summit of colon coagulated; g, formed, healed, and functioning stoma; h, functioning colostomy.

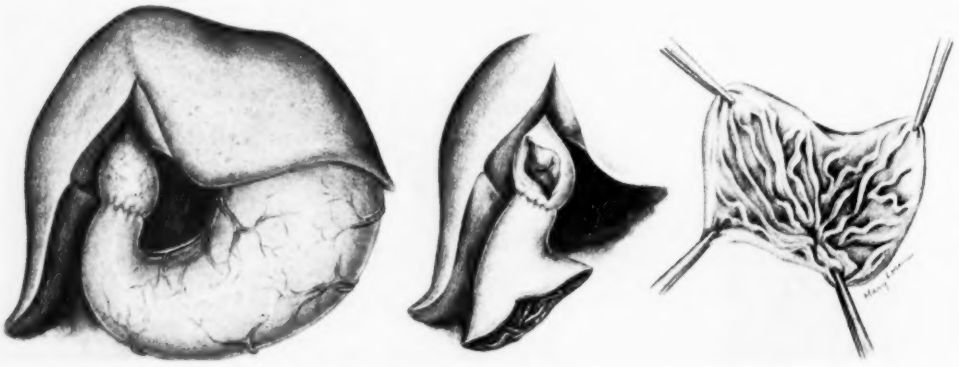

Fig. 6. Cholecystgastrostomy. a, Healed external appearance is shown; b, perfectly opened and functioning stoma looking into gall bladder; c, functioning stoma in pylorus.

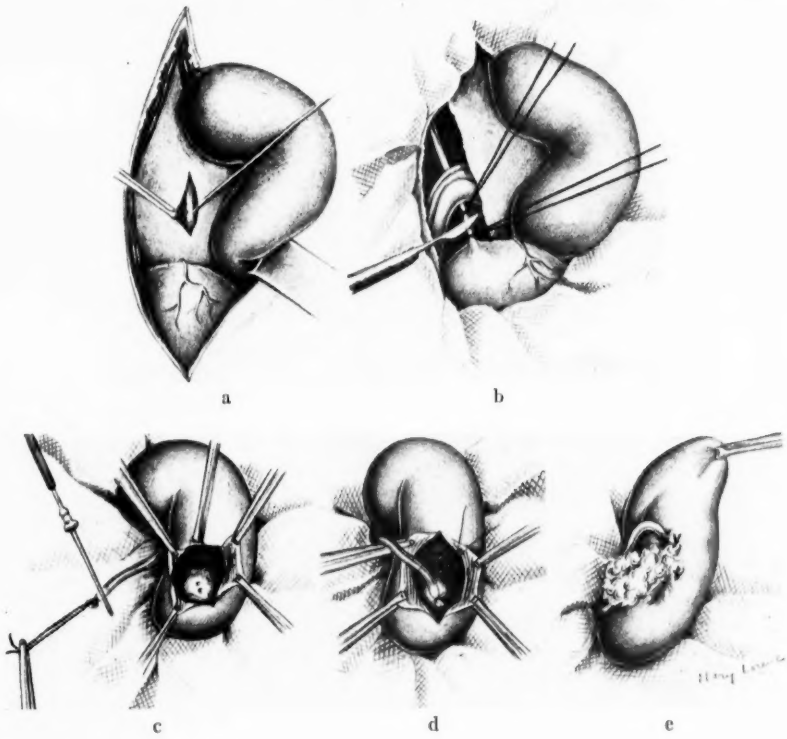

Fig. 7. Ureterocolostomy. a, Exposure of ureter, sigmoid exposed; b, division of ureter with ligation of divided ends; c, incision of sigmoid down to mucosa, coagulation of mucosa and ureter; d, opposition of coagulated areas of ureter and colon; e, suture of muscularis and serosa covered by omentum; f, external view of the anastomosis; g, the functioning valve openings of ureters in sigmoid.

toms or signs of intestinal obstruction. Four weeks after operation the dog was sacrificed and the perfectly formed and functioning anastomosis is shown in Figures 2, 3e, and 3d.

GASTRO-ENTEROSTOMY

The electrosurgical technique for this operation is identical to that for side-to-side anastomosis of the bowel. The multipolar elec-

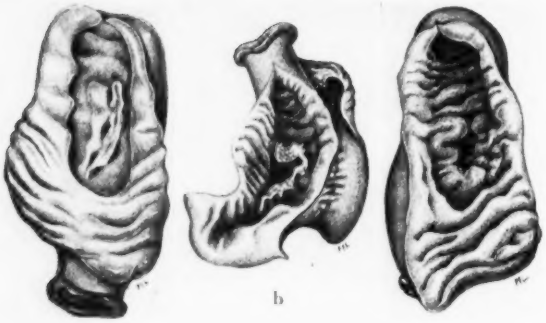

Fig. 8. Gross surgical pathology. a, Twenty-four hour appearance of stoma, with slough ready to separate; b, 48 hours slough separated; c, slough completely separated with early healing of mucosa.

trode is used with its prongs inserted deep into the muscularis of the stomach and 1500 milliampere current is used. The line of coagulation must be grey in color. In performing the line of coagulation on the loop of small intestine, 1000 milliampere current is used and the electrodes inserted, as described in the previous operation.

The illustrations, Figure 4, represent the results 10 days after operation of a gastro-jejunostomy as performed on the dog. The dog was allowed the usual rations and purposely no special postoperative care was taken, in order to give the operation the severest test possible. The animal made an uneventful recovery and did not miss a single meal.

Figure 4a represents the external appearance of the healed anastomosis and 4b the anastomosis opening perfectly formed, functioning, and well healed.

We do not agree with Ward, that the redundant mucous membrane of the stomach prevents the use of this technique. Failure to coagulate the mucosa properly is only a technical one and if the electrode is introduced as directed and coagulation done to a grey color with 1500 milliampere current, the mucous membrane will be properly coagulated.

This technique is not suggested to supplant the present technique by suture method. The operation has a place, however, when in repairing a ruptured duodenal ulcer a gastro-enterostomy is necessary. If suturing the ulcer causes a too marked stricture and a gastro-enterostomy is necessary, this technique should be used. The procedure is quickly done and does not expose the patient to spilling, hæmorrhage, or prolonged surgical technique. The anastomosis forms itself automatically, while the ulcer surface is healing.

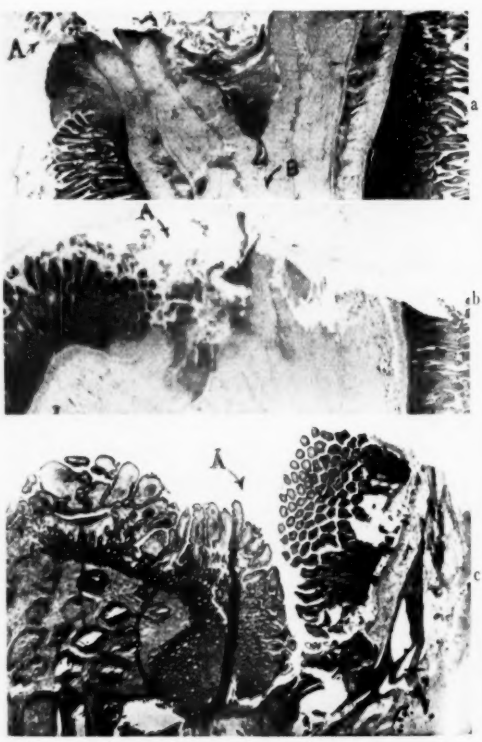

Fig. 9. Microscopic surgical pathology. a, Twenty-four hours postoperatively, slough still attached, *A;* b, 48 hours after operation, slough almost entirely separated, *A;* c, 8 days after operation, mucosa entirely joined and healed.

ENTERO-ENTEROSTOMY OF LARGE BOWEL TO LARGE BOWEL

A 6 inch loop of descending colon was sutured together near the mesenteric attachment by a continuous seroserous suture of 20 day No. 00 chromic (Fig. 5a). Two lines of coagulation with 1000 milliampere current and multipolar electrode were fashioned on each of the opposed loops as shown in Figure 5b. The lines of coagulation were opposed and sutured together by eight interrupted Lembert sutures of plain catgut No. 0 as shown in Figure 5e. The seroserous suture (Fig. 5d) was then completed, thus burying the line of coagulation. The colostomy was then sutured to the peritoneum and allowed to protrude from the skin wound. The summit of the colostomy was coagulated at one point with the bipolar electrode (Fig. 5f) so that in 36 hours it opened spontaneously. After 48 hours the colostomy functioned perfectly externally and internally. On introduction of a finger the anastomosis was found to be perfectly functioning. The dog moved his bowels both by rectum and through the colostomy. Figure 5g represents the internal view of the anastomosis perfectly formed and healed. Figure 5h shows the functioning opening of the colostomy on the abdominal surface.

Every surgeon knows the scrupulous asepsis necessary not only in performing anastomosis of the large bowel on the human, but also in animals. The danger of infection, leakage, and peritonitis in the face of the virulent flora is very great. The procedure as above described is perfectly adequate and safe and eliminates the difficulty of maintaining asepsis since the bowel is not opened.

This type of colostomy and anastomosis suggests two new uses. The first is a large pouch colostomy. Instead of the centripetal loop only emptying itself as in the conventional colostomy, we have a large reservoir of both loops. The centrifugal loop is turned into a centripetal loop. The large pouch allows of less frequent movement of the bowel and might afford more cleanliness and comfort to the patient.

The second use is the performing of what normally is the second stage of a Mickulicz operation in the first stage of that procedure. By the time the external colostomy is opened after the excision of the tumor, the internal anastomosis is already formed and functioning. This obviates the secondary clamping which is tedious and oftentimes difficult and unsatisfactory. We have performed such a procedure on the human with much success and gratification.

CASE REPORT

I. L., aged 56 years, was admitted to Bellevue Hospital, May 24, 1934, with a history of bloody stools, diarrhœa, and pain in the left iliac region since January, 1934. After a careful study including X-rays a definite diagnosis of carcinoma of sigmoid at its junction with the descending colon was made.

At operation the mass was found and a Mickulicz operation done. A 4 inch side-to-side anastomosis by electrocoagulation was performed just proximal to the mass as shown in Figure 5. Two days after operation, a finger introduced through the colostomy opening revealed the anastomosis already formed and functioning. On the third day, the patient had several small bowel movements by rectum and by the fourth day copious bowel movements.

In September, 1934, she returned to clinic and examination revealed a healed colostomy wound and a perfectly functioning sigmoid with normal daily bowel movements and a gain of 25 pounds in weight.

CHOLECYSTGASTROSTOMY

In performing cholecystgastrostomy by electrocoagulation the pylorus was selected for the anastomosis. As the pylorus is extremely thick in the dog, it offered an excellent test for the procedure.

The fundus of the gall bladder was sutured to the serosa of the pylorus transversely to its axis. Five millimeters away from this suture line and at its center, the gall bladder was coagulated at one single point with the bipolar electrode piercing only the serosa. The 1000 milliampere current was used and the coagulation continued to a light grey color and 5 millimeters in circumference. At a point in the pylorus directly opposite to the point of coagulation on the gall bladder a 5 millimeter point of coagulation was performed. The bipolar electrode was used, introduced well through the muscularis. The coagulation was performed to a grey color and 1500 milliampere current was used. The areas of coagulation were opposed by two interrupted sutures of 20 day No. 00 chromic catgut

passed into the serosa and muscularis to the right and left of the areas of coagulation. The primary seroserous suture was then completed so as to enclose completely the points of coagulation. Here again the dog made an uneventful recovery. Figure 6a represents the perfectly healed anastomosis externally without vestige of adhesions or leakage. Figure 6b is the functioning opening of the gall bladder into the stomach and Figure 6c the beautifully formed and healed opening of the stomach into the gall bladder. The same procedure may be utilized in anastomosis of the common biliary duct and biliary fistula to stomach or duodenum.

URETEROCOLONIC ANASTOMOSIS

As an experimental procedure, we attempted to anastomose the ureters to the large intestine of the dog by means of an electrocoagulation technique. The results were excellent and we hope that some urologist will reproduce the experiments and apply the procedure on the human.

The operation was done in 2 stages, 1 week apart. The technique and our observations and results will be described.

The sigmoid of the dog was identified and right ureter was exposed (Fig. 7a). The ureter was divided between clamps near the vesical termination and the ends ligated with chromic catgut (Fig. 7b).

A longitudinal incision 2.5 centimeters long was made on the serosa and muscularis of the sigmoid, thus exposing the submucosa. A point of coagulation 3 millimeters in diameter was performed on the submucosa near the lower angle of the longitudinal incision on the sigmoid. The unipolar electrode was pressed on the mucosa and coagulation performed with 1000 milliampere current to produce a light grey color. The inner aspect of the ureter was coagulated with the flat side of the electrode directly above the ligature. The coagulation was again carried to a light grey color with 1000 milliampere current and 2 millimeters in length. This is all shown in Figure 7c. By one interrupted suture (Fig. 7d) the end of the ureter was sutured to the submucosa opposing the points of coagulation. The muscularis and serosa were repaired over the ureter by interrupted sutures and reinforced with omentum (Fig. 7e).

The first stage was performed on the left side. When the second stage was performed on the right side we observed that the left kidney was slightly swollen and that the left ureter was four times its normal size but apparently emptying well. No leakage or infection was noted on the healed sigmoid surface of the anastomosis.

Two weeks after the second stage, the perfectly healthy dog was sacrificed and the autopsy revealed that the left ureter and kidney had returned to almost a gross normal. The right kidney was only slightly œdematous and the right ureter about twice its normal size (Fig. 7f). Both ureters were functioning well and the valve openings in the sigmoid are shown in Figure 7g. Gross and microscopic study of the kidneys showed that the left kidney was free of infection and normal. The right kidney showed slight cloudy swelling and a few pus cells in the urine from its pelvis.

The aseptic procedure is extremely simple and quick to perform and apparently very successful. Of necessity further studies and improvement of technique are necessary before it can be applied on the human.

SURGICAL PATHOLOGY

In order to determine exactly the time at which the anastomosis forms and the surgical pathology of the process, several anastomoses by electrocoagulation were performed and the specimens obtained at the end of 24, 48, 72 hours, and 8 days. The specimens were then studied grossly and microscopically.

Externally the 24 hour specimen revealed the seroserous sutures *in situ* with slight hæmorrhagic rings about the exit and entrance points in the serosa. These sutures were divided and it was found that the serosa of both loops between the suture line and the lines of coagulation had strongly agglutinated themselves. They could, with a little tension, be separated. There is no question, however, that with the sutures *in situ* and the agglutination of the serous surfaces no leakage could take place.

The coagulation necrosis of the coagulated area is shown in Figure 8a. The slough is still

in place except at certain points in the periphery where gas and liquid material could filter through. Slight traction on the slough easily separates it from the points of attachment.

The hæmorrhagic ring about the points of exit and entrance of the sutures in the serosa is rapidly disappearing in the 48 hour specimen. On division of the sutures and application of tension on the agglutinated seroserous surfaces, it was found that they could still be separated but with much difficulty. No leakage could possibly occur at this stage of the healing process. The slough (Fig. 8b) has completely separated with the exception of a few strands along the now perfectly formed and functioning anastomosis.

In 72 hours (Fig. 8c), the slough has completely separated even along the edges of the anastomotic opening and is being replaced by healthy granulation tissue.

The 8 day gross specimen reveals the mucous membrane in the anastomotic opening completely healed over.

Microscopically the 24 hour specimen shows the slough with the picture of coagulation necrosis (Fig. 9a, *A*). The slough is definitely still adherent but separating in places. The agglutinated seroserous surfaces (Fig. 9a, *B*) demonstrates the rather air-tight union of these two surfaces. The 48 hour microscopic section (Fig. 9b) demonstrates the separation of the slough, *A*. Along the periphery there are still some adherent strands of slough. The area of coagulation necrosis is definitely demarcated from the healthy intestinal wall. In 72 hours the remaining slough is completely separated.

In 8 days the mucosa is completely regenerated and healed from one side of the anastomotic opening to the other (Fig. 9c, *A*).

EVALUATION

We do not agree with Ward (2) that the electrocoagulation procedure cannot be relied upon on the stomach with its redundant mucosa. The failure is purely one of lack of properly performing the electrosurgical technique especially as to the depth and strength of the current and the degree of coagulation.

The technique of J. E. Briggs and L. R. Whitaker, calling for only one row of sutures, in our experience is adequate.

The attempt of W. W. Walker to perfect an aseptic anastomosis technique that would immediately open the loops at operation to our mind has no relation to the technique as it is described by J. E. Briggs, Ward, or ourselves.

In retrospect there are only extremely rare instances in which immediate anastomoses are necessary. In cases in which immediate intestinal drainage is necessary, temporary ileostomy, colostomy, or appendicostomy can be performed. In cases of intestinal resection, the anastomosis can be performed below the lumen of the resected bowel and the lumen allowed to drain externally while the anastomosis forms and begins to function.

The question most frequently asked is: "How does one know that the anastomosis will always form?" If the procedure is carried out as described, the anastomosis will always form. In a long series we never failed, even in our early experience with this method.

Secondary hæmorrhage is feared by many. We have never seen a case of secondary hæmorrhage in 15 consecutive operations by this method. All the animals were carefully watched after operation and at autopsy for this complication.

POSSIBLE APPLICATIONS TO THE HUMAN

1. Gastro-enterostomy—by electrocoagulation, when a such procedure is necessary in repairing a perforated duodenal ulcer complicated by stricture.

2. Lateral anastomosis after resection of segments of the bowel secondary to partial gangrene of a portion of the bowel as in strangulated Richter's hernia, gangrene due to bands, femoral hernia, etc. The two open ends of the loop left externally for drainage until the obstruction is relieved. The fistula can be then allowed to close spontaneously or repaired secondarily. This same procedure can be applied in performing an enterocolostomy.

3. A. Ileosigmoidostomy as a preliminary procedure to resection of the caecum.

B. Iliosigmoidostomy of choice with a temporary colostomy or appendicostomy.

4. Mickulicz operation completed in the first stage of the operation, eliminating the secondary clamping.
5. Cholecystgastrostomy or enterostomy.
6. Anastomosis of ureters to large bowel.
7. Without obstruction primary resection of large intestine with side-to-side anastomosis instead of the Mickulicz operation.

SUMMARY AND CONCLUSIONS

Fifteen consecutive and diversified anastomoses as performed on the dog and one as performed on man were successfully performed by electrocoagulation technique without mortality, leakage, infection, or hæmorrhage.

The procedure might in the future represent another step forward in surgical advancement.

We are deeply indebted to Dr. Leora McCloskey for her valuable assistance in the conduct of this work.

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THE ACTIVITY OF HAIR FOLLICLES WITH REFERENCE TO PREGNANCY

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THE condition of pregnancy is believed by many to influence hair growth in given regions of the body. However, there is no unanimity of opinion either as to the question whether growth is retarded or accelerated or with reference as to which areas are affected. Diverse observations have been made both on man and on some of the lower mammals.

As early as 1875, Slocum reported the case of a woman in whom hypertrichosis developed during each of three pregnancies, and *post partum* the hypertrichosis disappeared. Halban (1906) found quite regularly an increased growth of hair on the face of pregnant women, but this growth was more marked when the tendency toward hypertrichosis was already present. He also mentions an increased growth of lanugo hair over the whole body and especially in the region of the linea alba between the umbilicus and the pubic region; in each instance there was a return to normal *post partum*. In 1913, Harabath concluded that increased hair growth appears so regularly and typically in pregnant women that its appearance may be listed under the external symptoms of pregnancy. Many citations of cases of increased hair growth during pregnancy may be found in the literature, but there are also some cases reported of retarded hair growth coincidental with the period of gestation (Bechet, 1921; Ochs, 1928). These observations on women have been made for the most part by physicians who saw them as patients and had their attention called to the altered hair growth by the patient. In other words, the individuals were a selected group.

The reports on animals are of quite a different character, however, in that experiments were planned and carried out on unselected groups in which were included control series. Halban (1907) shaved the abdomen of female rabbits in a group half of which were pregnant. The hair regenerated much more quickly on the pregnant ones than on the control group. Harabath (1913) observed hypertrichosis in three pregnant dogs especially marked over the abdomen and to a less extent over the whole body. On the other hand, Dawson (1933) found a marked retardation of hair growth in pregnant guinea pigs particularly over the back, normal growth rate being restored *post partum*. Strangeways, later in the same year (1933), also observed these phenomena in guinea pigs.

Thus it appears that pregnancy retards hair growth in some species of the lower mammals and accelerates it in others. There is evidence that both effects are produced in man. The observations reported in this paper were made

in order to ascertain the effect of pregnancy on hair growth in certain selected regions of a group of unselected women.

Seven women, whose co-operation was obtained through the obstetrical clinic of Washington University, permitted weekly examinations for a period of approximately 9 months. The period was divided in each case into two parts by the birth of the child—thus, the examinations were made during the latter part of the pregnant period and for several months following delivery. The subjects, their ages, the number of the pregnancy in each case, and the lengths of the periods before and after delivery are shown in Table I.

TABLE I

Subject	Age (years)	Pregnancy	Weeks before delivery	Weeks after delivery
B	20	First	23	14
F	26	First	17	20
G	30	Second	12	24
M	24	First	25	11
Sa	26	Third	17	17
Sc	20	First	25	13
St	24	Second	25	8

The average length of the period over which each woman was examined was 36 weeks; 21 weeks before delivery and 15 weeks after delivery.

The color of the head hair was medium or dark brown in each case except that of M whose hair was red. The pubic, perineal, and lumbar regions were chosen for observation. The terminal hair of the pubic and perineal regions appears and acquires its distinguishing characteristics during puberty and is thus probably influenced by the changes occurring in the reproductive system at this time as much as, if not more than, hair on any other part of the body. (The axillary hair, of course, is in the same category as the hair of the pubic and perineal regions, but was not chosen for the experiment because of the quite prevalent custom of frequently shaving the arm pits). The lumbar region is covered with hair of the lanugo type which is typical of the general hairy covering of the body. In each region from ten to fifteen follicles were marked by injecting a small amount of India ink under the skin on either side of the follicle. These ink marks are permanent and therefore insure the identification of the follicle from week to week. Measurements of the hairs of the marked follicles were made weekly with a millimeter ruler.

The behavior of the follicle is known to be cyclic in character, i.e., a period of hair growth followed by a quiescent period after which the hair falls out and the growth period of the next hair is begun (Trotter, 1924). Thus, the measurement of the hairs made at regular intervals provides information on the rate of growth, the length of the active (or growth) period and the length of the quiescent (or rest) period of the cycle through which the follicle passes, and the ultimate length of the hair. Since all hairs in a given region are not in the same phase of the cycle at the same time, only part of the hairs at the time of the first measurement would be at the beginning of their growth period. The stage of the cycle of any individual hair was purely a matter of chance and not evident until after the second measurement. This resulted, therefore, in not having in every case full information on the growth and quiescent periods of the same hair, i.e., on the complete cycle of the follicle.

The hospital routine includes shaving the pubic and perineal regions just before delivery and whereas this proved to be a slight inconvenience from the standpoint of the experiment no information was lost. The hairs in the growing phase of the cycle continued to grow and this length added to the length of the hair before shaving gave the ultimate length, whereas those hairs in the resting phase of the cycle continued to remain quiescent and their cut ends which were on a level with the surface of the skin were plainly visible. The measurements on all the hairs in each region of each individual were averaged. The data are summarized in Table II.

A comparison of the rate of weekly growth before and after delivery in each of the three regions shows a slightly higher growth rate in some before delivery and in others after delivery. In no individual is the difference a marked one. On the whole, the tendency, although inappreciable and probably within the

range of error, is for a somewhat faster rate of growth after delivery than before. There does appear, however, a tendency for the hair of the perineal region to grow faster than the hair of the pubic region and for the hair of the lumbar region to grow the most slowly. There was no difference in the number of growing hairs during pregnancy and after delivery.

TABLE II.—AVERAGE MEASUREMENTS OF FOLLICULAR ACTIVITY IN THE PUBIC, PERINEAL AND LUMBAR REGIONS OF SEVEN WOMEN

	Subject	Rate of growth (mm. per week) before delivery	Rate of growth (mm. per week) after delivery	Growth period (weeks)	Rest period (weeks)	Ultimate length (mm.)
Pubic region	B	2.5	2.3	18	14	54
	F	3.2	3.2	15	15	51
	G	2.2	2.4	11	15	31
	M	2.8	2.6	14	12	38
	Sa	2.7	2.8	15	13	43
	Sc	2.7	2.8	14	13	43
	St	2.5	2.6	18	17	51
Perineal region	B	2.8	2.7	17	10	72
	F	3.0	2.8	15	10	55
	G	2.7	2.7	11	9	31
	M	3.0	3.6	23	12	75
	Sa	2.9	3.0	14	10	46
	Sc	2.7	2.6	13	10	50
	St	2.7	3.0	17	11	65
Lumbar region	B	1.9	1.4	6	11	10
	F	1.9	2.6	6	10	9
	G	2.0	2.0	5	9	7
	M			7	12	9
	Sa			5	12	10
	Sc			4	10	5
	St	1.0	1.4	6	11	8

The length of the growth period and of the quiescent period of the follicle and the total length of the hair are recorded without reference to the termination of pregnancy; this was essential because of the limited time over which the experiment was carried. Nine months is a relatively long period when the patience and time of the subject is considered, but a relatively short one when it is considered that a complete follicular cycle covers from 5 to 8 months. The growth period for the hair of the pubic and perineal regions is of longer duration than the quiescent period, but in the lumbar region it is approximately only half as long as the quiescent period. The quiescent periods of follicles of the lumbar and perineal regions are about the same length, whereas the quiescent period of the follicles of the pubic region is somewhat longer.

The lengths of the hair in the three regions are also variable. The perineal region has hair which is only slightly longer than the hair of the pubic region, but the hair of the lumbar region is very much shorter (approximately one-fifth as long) than either of these. Thus, it appears that the shorter hair (lumbar) has a much longer quiescent period than growth period and that the longer hair (pubic and perineal) has a somewhat longer growth period than quiescent period—of these two regions the perineal hair is the longer with a definitely longer growth period than rest whereas the pubic hair which is not quite so long as the perineal has growth and quiescent periods of more nearly equal length. It may be noted also that the lengths of the individual growth periods of the pubic and perineal regions are approximately equal (exactly equal in the case of B, F, and G) with the exception of M whose perineal hair grows for a much longer period than her pubic hair. The similarity in length of the growth periods of the two regions in the 6 individuals is further evidence that the excessive length of the hairs of the perineal region is due to a faster rate of growth. However, the number of subjects is too small to warrant either stating an average for the group or calculating a correlation between two or more of the observations recorded.

It may be assumed that the rate of weekly growth times the length of the growth period which is given in weeks should indicate the exact total length of the hair. This does not always follow and the discrepancy is explained by the fact that the rate of growth data are compilations of partial cycles of follicular activity (the weekly rate of growth could be determined from measurements of length for only a few consecutive weeks of the growth period), also the ultimate length of the hair could be determined after the quiescent period was recognized which necessitated only a few

weeks, whereas the length of the growth period could only be determined from follicles which had been observed from the time the hair appeared until the quiescent phase was established. Thus, many more hairs contributed information on rate of growth and on ultimate length of the hair than on the length of the growth or of the rest periods, both requiring observations throughout the entire period. The discrepancy, therefore, is the result of individual variation of the hairs.

The amount of hair in the regions under observation did not change perceptibly during the entire period of 9 months. Nor could any one of the subjects notice alteration in any part of the hairy covering of her body.

SUMMARY

Measurements on the rate of hair growth in the pubic, perineal, and lumbar regions of a group of seven women made during the latter months of pregnancy and the first several months following delivery showed no variations which could be attributed to the pregnant condition. The duration of the growth period was longer for the longer hairs (pubic and perineal) than for the shorter hairs (lumbar). The quiescent period of the follicular cycle was longer than the growth period in the lumbar region, considerably shorter than the growth period in the perineal region, and only inappreciably shorter in the pubic region.

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CLINICAL SURGERY

FROM THE STANFORD SURGICAL SERVICE

AN OPERATION FOR TUBERCULOUS EMPYEMA

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THE ordinary form of rib resection with introduction of a drainage tube is notoriously unsatisfactory in tuberculous empyema, especially in empyemas the pus of which contains either tubercle bacilli alone or no demonstrable organism at all; i.e., empyemas not secondarily infected. So called "closed" drainage, drainage by means of a tube introduced into an intercostal space through a trocar, is equally bad. Both of these methods always lead to secondary infection and almost always to fever, sepsis, and death.

Some forms of tuberculous empyema, however, occasionally purely tuberculous ones, more often secondarily infected ones, produce such consistently high swinging temperatures and such severe symptoms of toxicity, that surgeons are impelled again and again to drain in spite of previous warnings and in spite of full knowledge of the trouble that usually ensues.

Several methods of treatment are proposed for such toxic tuberculous empyemas. Aspiration with a syringe, replacement of the pus by an equal quantity of air, and irrigation of the pyopneumothorax cavity with countless varieties of antiseptic solutions have been recommended. Aspiration often results in abatement of fever and sepsis for a few days and even weeks, but usually needs be repeated so often and so long that both the doctor's and the sufferer's patience are exhausted. At times it leads to cure, usually not.

Oleothorax (injection of gomenol and various other antiseptic or aseptic oils) has led to good results in some hands (Matson); to disastrous results in others.

Extensive thoracoplasty may succeed in obliterating the empyema cavity, but the operation needs be extensive and formidable. It entails considerable risk to patients weakened by long toxicity and considerable crippling at best.

Cure of these tuberculous empyemas is made difficult by the tenacity with which the underlying lung resists expansion. It is also made difficult by the unfavorable effect of an inlying drainage tube of any kind. The presence of the tube itself, I think, tends to keep up fever and sepsis. Some tuberculous empyemas treated ill advisedly by rib resection and drainage heal if one does nothing more than remove the drainage tube.

A desire to obviate a drainage tube led to an operation which has proved of use in a number of secondarily infected tuberculous empyemas. Encouraged by its success it has with some hesitancy recently been used in a few obstinately toxic tuberculous empyemas, in which no pus forming organisms could be found.

The operation causes the underlying lung to expand; it is, therefore, *not applicable* to those empyemas in which the lung is so badly affected that expansion to any degree seems inadvisable. The question of applicability must be decided by clinical pulmonary symptoms and especially by X-rays taken prior to the appearance of the complicating empyema. If cough and expectoration of bacillus bearing sputum still persist and if early roentgenograms reveal large cavities or other extensive parenchymal damage, some form of thoracoplasty is probably safer. Fortunately, in many empyemas the lung seems little if at all affected and partial if not total expansion seems allowable. If but partial expansion seems advisable the operation to be described should be complemented by an upper thoracoplasty.

TECHNIQUE

Under local anæsthesia a U-shaped flap of skin and subcutis is outlined about half way between the posterior axillary line and the line of the inferior scapular angle. The flap has a base about 2 inches wide which lies about one rib higher than the bottom of the empyema cavity, so that the rising diaphragm may not stop drainage; it is about 2½ inches long, the length of two ribs and

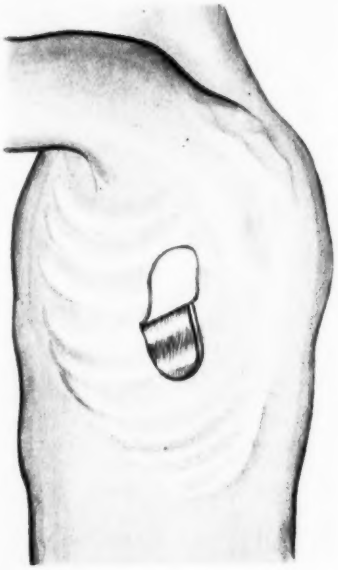

Fig. 1. Flap of skin outlined.

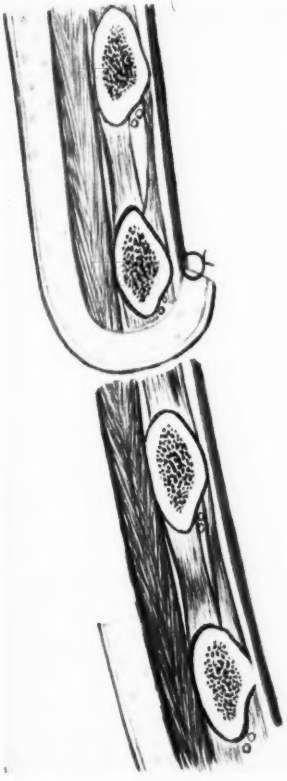

Fig. 2. Cross section of chest wall showing skin flap turned into chest and attached to pleura.

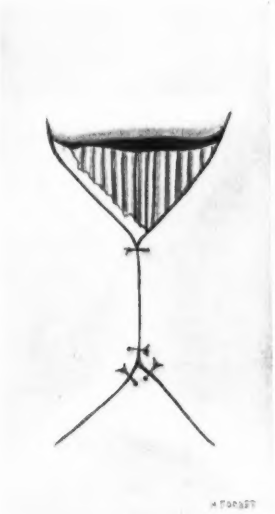

Fig. 3. Skin flap turned into chest cavity; edges of defect approximated by sutures.

their intercostal spaces, long enough to reach into the pleural cavity without the least tension and longer, therefore, in fat patients than in lean ones. The rib underlying the top of the flap is resected, the amount resected equalling the width of the flap. If the flap is too narrow and the resection too scant, drainage will be insufficient. It is good, I think, to strip the rib with a cautery instead of a raspatory and to inject the bared intercostal nerve with 1 cubic centimeter of absolute alcohol. The tip of this flap is turned into the chest and tacked to the pleura with one or two chromic catgut stitches; the edges of the defect in the skin are approximated with a few stitches of silkworm.

This thoracotomy needs no tube, for the skin flap which lies against the soft parts of the chest keeps the wound open. It remains open until the lung reaches the chest wall, after which it spontaneously and automatically closes without further ado. It has a valve action; each cough or rise in intrapulmonary air pressure expels a little air from the thorax and causes a gradually increasing negative pressure in the empyema cavity, for it is more difficult for air to enter through this valve-like wound than to escape from it. This fact is readily demonstrable (although in theory the valve might seem to work the other way) if the wound is opened by an instrument at a change of dressings, when air will enter it with a sucking sound. If much negative pressure is desired, overzealous and overcleanly nurses should be cautioned not to change the dressings or to content themselves with changing the outer ones only and to leave the inner ones in place as long as possible —for many days.

This operation, it will be seen, differs entirely both in theory and practice from the valve recently proposed by Nichol for acute empyema. Details, with X-rays, case histories, and other data will soon be published.[1]

[1] Western J. Surg.

BIRTH FRACTURES OF THE FEMUR

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THE problem of caring for fractures of the newborn, the result of birth injuries, occupies a rather unique position in medical science in that it concerns no particular specialty. This fact at once becomes evident when search is made in the literature; reference must be made not only to the obstetric and pediatric, but also to the orthopedic and surgical, literature. It is surprising, moreover, how little has been written on the subject, at least in so far as data regarding the final results are concerned.

For the purpose of acquiring a wider knowledge of the entire problem, study has been made of the fracture material found at the Obstetrical Clinic in Lund during the years 1900–1934. It is the preliminary report of this investigation that I present in this paper. The present report concerns fractures of the femur only.

During the study three main points were kept especially in mind: (1) the immediate process of healing; (2) the prognosis, especially with respect to the end-result; (3) the best treatment.

Only brief mention will be made of our findings regarding these points as brought out in our study of the literature.

As to the immediate process of healing, it is well known that the healing tendency in infants is great and that even a severe displacement can be remolded into an anatomically good result through the forces of nature (v. Reuss, Naujoks).

As to the final results, the accounts given in the literature are very few. As a rule, however, it is evident that the favorable result of primary union has been taken as a warranty that the final result will also be good. A few isolated reports of poor results, especially regarding resultant shortenings, have been made (Staffier, Krukenberg, Naujoks). In these cases, however, the time of final examination was not more than 10 years after birth.

The question of treatment has received more attention in the literature than the two other points in question, and a number of different methods have been recommended. Up to the present time, however, no definite agreement has been arrived at as to which is the best method. Generally speaking, the methods proposed may be classified into two main types: fixation without extension on the body (Credé, Kuestner, Dollinger, and others), by means of plaster of Paris (Dollinger), with splints (Isbister, Spitzy); and extension in some form (Schede, Riese, Jones, Oeri, Schanz, Gordon, and others). Of these two different types of treatment, the former, that is, simple fixation without extension, may be said to be employed in more cases than is the extension method.

AUTHOR'S STUDY OF CASES

My own study of the records of the fractures occurring at the Obstetrical Clinic in Lund shows that in approximately 40,000 deliveries there were 16 fractures of the femur. All of these fractures, except 2, occurred in breech presentations. Of the two exceptions one occurred in delivery by cæsarean section, the other in the manual bringing down of an extended leg. The oldest fracture dates from 1914.

With reference to the first point, as to the immediate process of healing, an examination of the 13 remaining fractures (3 infants died during birth) confirms the statements made in the literature as regards rapid healing. Union of the fractured bones is so rapid that, on the basis of weeks as a suitable unit of time in the treatment of fractures in adults, we must reckon with days in infants—soft callus appears even in a few days, and bony callus has been observed even in the first week (Bors). This emphasizes the fact that definite reposition should be completed within this time; otherwise the opportunity of obtaining a satisfactory position will be lost, at least lateral dislocations and shortenings will then be difficult to correct. Indeed, the majority of reports of our cases, as they now appear in the records and in the roentgenogram, bear witness to the difficulties encountered, for it is evident that in spite of repeated attempts at reposition and fixation the fractures were usually left to unite in an anatomically bad position.

The most common malposition is, as appears from the illustrations, medial displacement and flexion of the proximal fragment, this malposition being produced by the contraction of the flexors and adductors inserted on this fragment. Shortening is also a usual finding and is generally marked; in certain cases it may be at least one-fourth of the length of the femur (Fig. 6).

Osseous union occurred, however, in all cases and sufficient firmness was acquired within 14 days. In those cases in which the process of healing was followed by means of radiographic exam-

Fig. 1, left. Fresh fracture of the femur. The three following illustrations show the rapid healing and spontaneous correction.

Fig. 2. The same fracture after 3 weeks. Large callus. No better position could be obtained. Extension was not tried.

Fig. 3, left. The same fracture after 7 weeks. Spontaneous correction has commenced.

Fig. 4. The same fracture after 10 months. The position is good.

inations for somewhat longer periods after fracture, it was evident how wonderfully even severe malpositions were modified and gradually assumed a position quite satisfactory anatomically. It should be noted that this process of remolding has taken place for the most part during the first 6 months of life and not, as might be expected, after the child had commenced to stand on its legs (Figs. 1–4). Hence, there is no need of weights to guide the spontaneous correction of the malposed member; the normal tonus and play of the musculature seem to be sufficient for this purpose.

Thus, in spite of the fact that in general the fractures were allowed to unite in an anatomically unsatisfactory position, osseous union always did occur; second, the malposition was spontaneously reduced in a rather short time. As we shall bring out later, no inconvenience has been experienced and no suffering has been noted in the early period of growth. The result as to primary healing can therefore quite justly be characterized as good.

In order to obtain an idea as to the final result in these cases, follow-up examinations of the 6 oldest fractures of the femur were made. A brief summary of these 6 cases follows:

Case 1. No. 495/1924. Male child had a fracture of the left femur following breech presentation. Labor was long and extraction was accomplished by fillet. The fracture was fixed in various ways for a period of 3 weeks. The result was healing in bad position. Healing was normal and patient learned to walk when 16 months old. He has never had any subjective trouble. In 1933, Dr. Brandberg reported that the boy was then 9 years of age and the roentgenogram showed a marked, even, forward curvature of the upper part of the femur. The left leg was 0.5 centimeter shorter than the right. A certain forward curvature was also visible in the upper part of the thigh (Figs. 5–7).

Case 2. No. 907/1919. A female infant received a fracture of the left femur during prolonged labor, breech presentation, and extraction by fillet. Fixation along the trunk was maintained for 14 days. Primary position of fragments was bad. The process of healing was normal. Patient learned to walk in the usual time and had no subjective trouble until the last 2 years when she began to notice that the left leg was weaker than the right but the weakness was so slight that it was hardly noticeable. In 1934, when she was 15 years old, an X-ray examination showed only a slight trace of the fracture. Both legs were of equal length. The left thigh was 1.5 centimeters smaller around than the right thigh; the left calf was 0.5 centimeter smaller. A scar, the result of the application of the fillet, was noticeable in the medial part of the groin (Figs. 8–10).

Case 3. No. 418/1919. A male infant, in breech presentation, received a fracture of the left femur during delivery. The mother was febrile and extraction was accomplished by fillet. Fixation of the fracture was maintained for 18 days. No roentgenogram was taken. Normal union took place and patient learned to walk at the usual time. He has never had any subjective symptoms. In 1934, when he was 15 years old, examination showed no more than a trace of the fracture. The legs were of equal length. In circumference the left leg measured 0.5 centimeter less than the

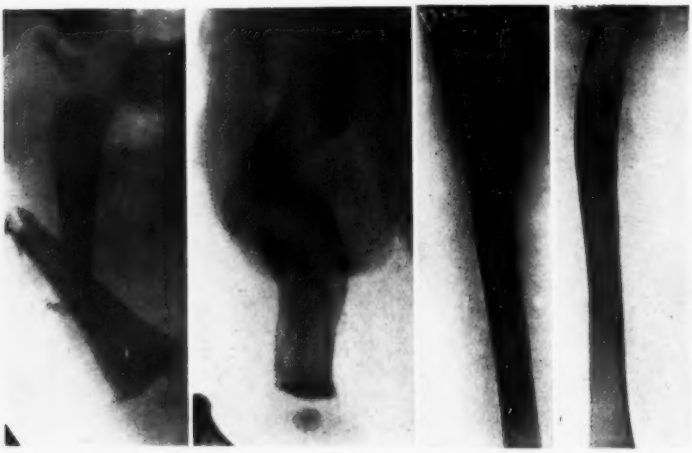

Fig. 5. Fig. 6. Fig. 7.

Fig. 5. Fresh fracture in a bad position, Case 1.

Fig. 6. The same fracture as in Figure 5, after 5 weeks. Large callus. Very bad position. No extension. Case 1.

Fig. 7. The same fracture as shown in Figures 5 and 6 after 9 years. There is still marked curvature of the femur Case 1.

right. A plainly visible scar was noted in the groin, the result of the application of the fillet. The ability to extend the leg was somewhat limited in both hip joints. Marked lumbar lordosis with pelvis projecting backward was noted (Figs. 11–12).

Case 4. No. 504/1914. A male infant received a fracture of the left femur during delivery. The child was in breech presentation and threatening asphyxia prompted extraction by fillet. Fixation on trunk was maintained for 3 weeks. Primary position was bad. Healing proceeded normally. Patient learned to walk in the usual time and had no trouble during childhood. He began to notice symptoms at the age of 15 or 16 years but even though pain increased slightly it was only moderate at all times. He walked with a raised heel. In 1934, at 20 years of age, the roentgenogram showed but very slight trace of the old fracture. The left leg was almost 2 centimeters shorter than the right; at the thigh it was 2 centimeters more around. The pelvis was oblique (Figs. 13–15).

Case 5. No. 958/1914. A female child, in breech presentation suffered a fracture of the left femur during delivery. Labor was atonic. Extraction was accomplished by fillet. Fixation to trunk was maintained for 18 days. Primary position was bad. Union took place normally and patient

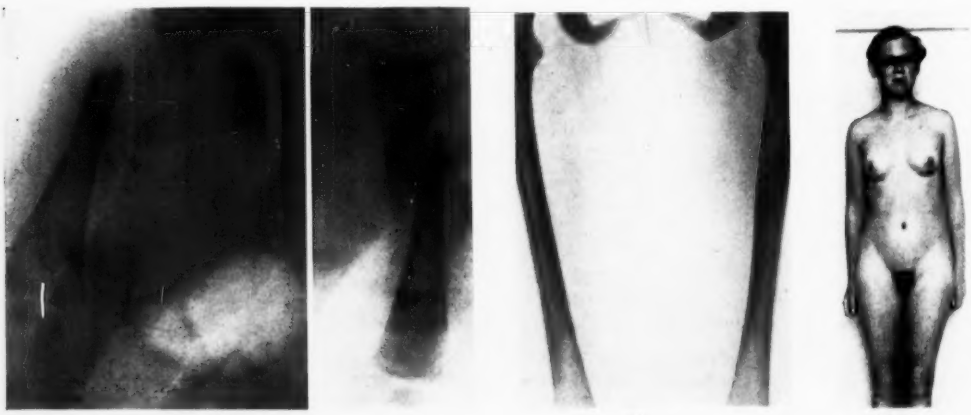

Fig. 8. Fig. 9. Fig. 10.

Fig. 8. Fracture of the femur. Marked callus already. Bad position. No extension. Case 2.

Fig. 9. The same fracture as in Figure 8 after 15 years. Now only a slight thickening of the femur remains. Case

Fig. 10. The same case as in Figures 8 and 9. The fillet has left a scar in the medial part of the left groin. Case

Fig. 18.

Fig. 19.

Fig. 18. Fresh, 6 day, fracture of the femur. No extension. Case 6.

Fig. 19. Patient now 20 years old. Fracture is scarcely visible. There is, however, a shortening of 2 centimeters. Case 6.

Fig. 19a.

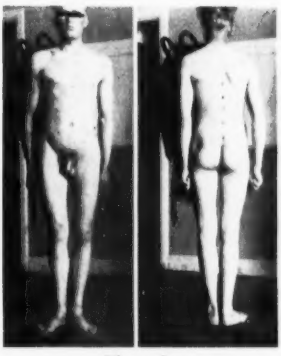

Fig. 19b.

Fig. 19a. Compensatory scoliosis with convexity to the side of the shortened right leg. Case 6.

Fig. 19b. The same case as in Figures 18 and 19. Shortening of the right leg. The pelvis is inclined with spina iliaca anterior superior and the gluteal fold lower on the right side than on the left. Patient has a scoliosis with the convexity to the right. There is a deep impression present in the right groin which has been left by the fillet. Case 6.

the trouble did not manifest itself until several years after birth. This may signify that such difficulty will increase as the patient grows older; and too the possibility cannot be excluded that the patients who are at present free from symptoms will later on in life develop symptoms of static insufficiency. Thus, we should not allow ourselves to be deceived by the beautiful roentgenograms obtained in the following examinations but rather bear in mind that in a clinical sense the result may not always be entirely in accord with the X-ray picture.

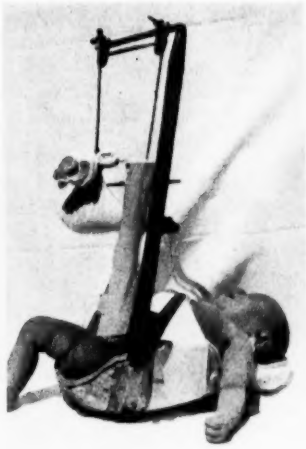

Fig. 20.

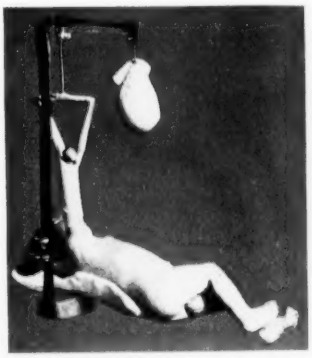

Fig. 21.

Fig. 22.

Fig. 20. The extension apparatus applied for a fracture of the femur.
Fig. 21. The extension apparatus applied for a fracture of the humerus.
Fig. 22. Nursing is not disturbed.

Fig. 23.

Fig. 24.

Fig. 23, left. Fresh fracture of the femur.
Fig. 24. The same patient as in Figure 23, after 26 days. Treated in the extension apparatus. The only case without shortening.

Fig. 25. The same case as in Figures 23 and 24 after [illegible] months.

TREATMENT

From what has been said it is evident that the treatment must aim at a definite result so that even the ultimate result will be entirely satisfactory. This point merits special emphasis since the difficulties encountered in treating fractures in these little patients, where the primary union is almost always good, may otherwise encourage carelessness.

The treatment employed up to last year at this clinic was fixation of the fractured leg close to the body with the foot at one of the shoulders, sometimes with, and sometimes without, wooden splints. In exceptional cases plaster of Paris or metal splints bent at right angles have been used (Spitzy). The period of fixation has in general been 2 to 3 weeks. The patients have then been discharged and no further treatment has been given. Extension treatment was not employed previously but was introduced last year. The advantages of this latter method of treatment have, however, been so great that it should undoubtedly be preferred to simple fixation. Shortening, perhaps the most important malposition, cannot possibly be avoided but by extension (Fig. 24).

The type of extension employed at this clinic was first advanced by Schanz (1928) and later on also described by Obadalek (1933). The principle of this method is to make extension possible without having to fix the child to an uncomfortable cot, which is rather difficult to move about. With this method the child can be easily carried about with the entire extension apparatus applied. Similar methods have been previously mentioned by Boorstein, Gordon, and others.

Of the older methods the simple fixation treatment, with or without splints, also possessed the same advantage that it allowed the child to be carried round, but it had one drawback in that it frequently gave an unsatisfactory reduction of the fracture and required almost daily dressing. The extension treatment in which the extension apparatus was attached to a bed (Schede) had the advantage of giving a better reducing result but it was inconvenient, for one thing because it rendered nursing difficult.

The new method of extension introduced by Schanz combines the advantages of the two older methods mentioned, without possessing any of their disadvantages. The technical arrangements are simple as may be seen from the pictures (Figs. 20–21). On a bed of plaster of Paris an extension gallows made of iron is fixed on a level with the hip joints. This device may be jointed at the base. A new plaster-of-paris bed can be made for each separate case, the extension gallows being adjusted to the hip of the child, or to one of its shoulder joints in the case of a fractured humerus. If desirable, the surgeon may have four such beds in readiness, one for each extremity. The apparatus can easily be made by any blacksmith or ordered from some firm of instrument makers. The application of the extension device to the fractured limb can be performed by means of zinc glue or mastisol but the simplest way is to use a good quality of common adhesive plaster.

The weight extension is generally made so heavy that the lower part of the patient's body is lifted a little from the plaster-of-paris bed. The patient is then suitably held to the bed by means

of a wide bandage. The period of fixation varies from 2 to 3 weeks.

Although the entire arrangement may perhaps appear rather complicated, it is in fact very easy to superintend during the course of treatment. If the apparatus is ready for use the work of attaching the child to it can be accomplished in a very short time. That it fixes the fracture in a firmer manner than by fixing the limb on the body is evidenced by the fact that the child's crying is considerably less when the leg is extended. Another advantage is that, once the extension bed has been applied to the fracture, there is no need of repeated bandaging or so much supervision as in the majority of other methods of fixation. A fracture once reduced can be easily kept in position by this method. Finally, it does not in any way hinder nursing (Fig. 22). Its main advantage, however, is that it insures a better reduction than any other treatment without extension.

SUMMARY

An investigation has been made of the records of birth fractures at the Obstetrical Clinic at Lund during the years 1900–1934. During this period 16 fractures of the femur occurred. Three children died in connection with delivery. The 13 remaining were examined.

1. Reposition was always difficult. The fractures were frequently left to unite in an anatomically unsatisfactory position. Osseous union has always occurred, however, in a short time. The mal-union has been reduced by a spontaneous remolding into a satisfactory position in the course of the following months. No trouble has been experienced by the patients during the period of growth. The children all learned to walk in the usual time. The *primary healing result* has therefore been good.

2. The examination of the *final results* in the 6 oldest patients, at present from 9 to 20 years of age, has shown that in 1 case there still remains a marked curvature of the femur at the site of the old fracture, whereas in the 5 other cases hardly any trace of the fracture can at present be seen in the roentgenogram. In several cases, however, various changes were observable, such as, subjective trouble and shortening. These changes were, indeed, very slight but they cannot be denied every importance.

3. The best *method of treatment* has been found to be extension, the method employed being extension in a portable plaster-of-paris bed (Schanz, 1928). Reduction should be completed after the first few days or at any rate within a week of the occurrence of the fracture. Otherwise it will be rendered more difficult on account of the early formation of callus.

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PERIPHERAL NERVE BLOCK IN OBLITERATIVE VASCULAR DISEASE OF THE LOWER EXTREMITY

Further Experience with Alcohol Injection or Crushing of Sensory Nerves of Lower Leg

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FOUR years ago we reported a technique for alcohol injection of the sensory nerves of the lower extremity (4) in order to relieve the intense rest pain in cases of advanced obliterative vascular disease. At that time our series consisted of 11 cases and the immediate results were gratifying both to us and to the patients. Since then we have employed this procedure in over 50 patients. Sufficient time has elapsed to report our results in the first 45 cases, and to state our present feeling in regard to the value of this operation not only in relieving pain but also as an effective method of treating the disease.

CASES SUITABLE FOR OPERATION

The cases may be divided into two groups according to the pathological process in the vessels (Table I). Twenty-eight were classified under the diagnosis of thrombo-angiitis obliterans, and 17 as arteriosclerosis. We wish to emphasize that the patients in this series are suffering from an advanced stage of the disease, with extensive obliteration of main vessels, with severe rest pain, and in nearly every case with ulceration or extensive gangrene. They are not the early cases which usually respond to systematic hygiene, postural exercises, rest, forced fluids, and elimination or curtailment of the use of tobacco, as has been previously described (1). These are cases which for the most part have had a trial of conservative treatment in the hospital for 1 to 2 weeks before resorting to this operation.

ANATOMY AND TECHNIQUE

The anatomy of the lower leg and the technique of operation were described in detail in our first article (4). This will be repeated briefly to emphasize certain points which we believe to be important. Figure 1 shows the position of the five nerves supplying sensation to the foot. It may be necessary to block one or all of these nerves in a given case. They are exposed through small vertical incisions in the middle or upper thirds of the lower legs, under local anæsthesia. Only one nerve should be blocked at a time. The others can be done at intervals of a few days to a week. We believe that too extensive an operation at one sitting may result in rapid advancement of gangrene in the foot, as occurred in one of our first cases. The operation should be done with the greatest care and asepsis. It is essential to know exactly where the nerve in question lies. This knowledge can be acquired only by careful dissection of amputated limbs, and nobody should attempt this operation until he is thoroughly familiar with the course of the nerves. There should be no lateral dissection of the layers. One should be able to place the skin incision exactly over the nerve, avoiding intramuscular fascial septa where small collateral arteries are prone to run. Tooth forceps, rake retractors, and such coarse instruments should never be used. In these advanced cases it is almost never necessary to use a hæmostat. There are no spurting vessels if the incisions are properly placed, and the capillary oozing in the fat layer can readily be controlled by gentle pressure with a gauze sponge. When it is necessary to incise the deep fascial layer, we do not advise suturing it. Only the finest of plain catgut ties (No. 000) are used when necessary. Silk sutures are placed in the skin without the aid of forceps. Perfect approximation is essential, and sutures should be tied loosely. Operation should never be done in the presence of lymphangitis. Incisions should never be made near areas of migrating phlebitis.

METHOD OF BLOCKING THE NERVE

In the earlier cases (3) the nerve was carefully freed and the surrounding tissues protected by moist gauze. A segment of the nerve, defined between two snug catgut ties, was then distended with 95 per cent alcohol (Fig. 2). The object of this technique was to localize the extent of nerve injected so that the time of regeneration could be controlled and to prevent irregular diffusion of alcohol along the nerve. This we felt was sometimes followed by paræsthesias due to incomplete necrosis of nerve fibers at the upper limit of alcohol diffusion. In the more recent cases we have simply crushed the nerve with an instrument over an extent of ¼ inch if rapid regeneration was

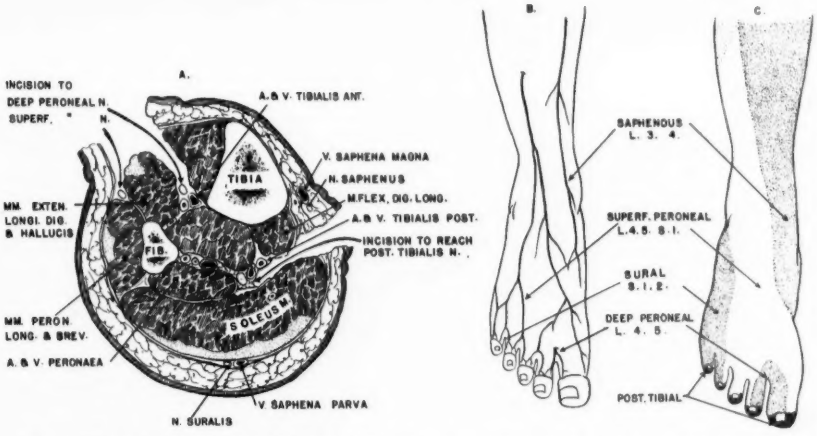

Fig. 1. Showing the anatomical position of the five nerves supplying sensation to the foot and the areas supplied by each. They can be blocked through small vertical incisions in the middle and upper thirds of the lower legs without causing important motor paralysis. Adapted from John Homans' *Textbook of Surgery*, 1932.

desired, or up to ½ inch if prolonged anæsthesia seemed necessary. We favor this latter technique at present because it can be done through a shorter incision, takes less time, and avoids the danger of spilling alcohol into the tissues. Crushed nerves regenerate more rapidly and apparently as completely as those injected with alcohol. A nerve crushed over an extent of ¼ inch will regenerate in about 3 months. We do not favor actual section and suture of the nerve, as has been advocated by Laskey and Silbert, because this would appear to make the operation unnecessarily complicated and difficult, although it would, of course, be equally effective.

COMPLICATIONS

Most of the complications which follow this operation are the result of faulty technique. Among those which we have encountered are delayed healing of the incision; infection in the wound; ulceration of the anæsthetic skin as a result of improper weight bearing and unnecessary pressure from poorly fitting shoes; precipitation of gangrene of the foot following too extensive an operation at one sitting.

All of these we have practically eliminated by improvement in technique and careful attention to weight bearing and proper shoes after operation. If the deep peroneal nerve is blocked in the upper third of the leg, paralysis of the extensor hallucis longus may result. In this case an ulcer may develop on the plantar surface of the great toe due to improper weight bearing because of toe drop. Amputation of this toe may be necessary. The intrinsic muscles of the foot are temporarily paralyzed following block of the posterior tibial nerve. This will be followed by atrophy of the muscles concerned, which as far as we know has never resulted in any important loss of function.

BENEFICIAL SEQUELÆ

Besides the elimination of pain, which alone is sufficient indication for the operation, other important changes take place. First of all, the general condition of the patient improves. He is able to sleep, eat, and carry out other conservative measures such as postural exercises. He is able to curtail or eliminate his tobacco. Locally, infection can better be controlled by frequent antiseptic dressings, slough can be trimmed away, and the healing of the wound is greatly accelerated.

The circulation to the part may actually be materially increased because the anæsthetic area is completely sympathectomized. Certain of these patients have a large element of vascular spasm as well as obliteration. As the efferent vasoconstrictor sympathetic fibers lie intermingled with the sensory components of the peripheral nerves, they likewise are blocked. The anæsthetic area is therefore sympathectomized and the surface temperature may be elevated as much as 15 degrees F. in some cases (Fig. 3). This, of course, is temporary and as sensation returns, vascular spasm returns. If the surface temperature rise has been marked, this operation

TABLE I.*—THROMBO-ANGIITIS OBLITERANS

Case No. Hosp. No.	Age		Local lesion	Peripheral vessels Pop.	D.P.	P.T.	Nerves blocked	Minor amputation	Major amputation	End-result
1. 291495	39		Great toe amputated for gangrene. Sepsis and severe pain in stump	o	o	o	Posterior tibial	Great toe		Slow but painless healing of disarticulated toe in 2 months
2. 287408	36	*Entry I*	Ulceration of tip of right great toe	o	o	o	Cutaneous branch of peroneal, posterior tibial, right	Right great toe		Terminal phalanx removed, pain relieved, but infection extended along flexor tendon sheath as far as os calcis. Toe amputated
		E. II	Painful unhealed amputation stump right great toe	o	o	o	Posterior tibial, superficial and deep peroneal, right		Gritti-Stokes, right	Pain not relieved because gangrene extended beyond anæsthetic area, Gritti-Stokes amputation
		E. III	Severe pain and discoloration in toes of left foot	o	o	o	Superficial and deep peroneal, posterior tibial, left		Gritti-Stokes, left	Pain only partially relieved because gangrene extended into lower leg, Gritti-Stokes amputation
3. 295220	36	*Entry I*	Continued pain in foot after amputation of second and third right toes for gangrene	+	o	o	Cutaneous branch of superficial peroneal, deep peroneal, and posterior tibial	Second and third toes, right		Complete relief of pain. Healed in 2 months
		E. II	Painful gangrene left great toe	+	o	o	Superficial and deep peroneal, posterior tibial	Left great toe		Toe amputated. Stump failed to heal, pain relieved but reappeared
		E. III	Discharging sinus in amputation stump left great toe	+	o	o	Superficial and deep peroneal, posterior tibial, saphenous, and sural	Second, third, and fourth toes left		Pain not relieved and healing incomplete
		E. IV	Left foot stump unhealed and infected	+	o	o	Superficial peroneal, sural, and saphenous	Heads of all metatarsals		Complete relief of pain. Healed slowly with skin graft, 8 months after injection
4. 280896	43		Painful ulcer of great toe	+	o	o	Posterior tibial, deep peroneal, and cutaneous peroneal		Gritti-Stokes	Final amputation, as ulcers did not heal
5. 298991	37		Painful ulcers dorsum of foot and great toe	o	o	o	Posterior tibial, cutaneous peroneal, and sural			Pain relieved, ulcers grafted
6. 314170	48		Painful ulcer of great toe	+	o	o	Sural, posterior tibial, and superficial peroneal		Gritti-Stokes	Ulcer failed to heal. Gritti-Stokes amputation
7. 297047	46	*E. I*	Painful ulcer right second toe	o	o	o	Superficial and deep peroneal, posterior tibial			Pain relieved. Tip of toe healing on discharge
		Entry II	Trophic ulcer great toe from toe drop and faulty weight bearing	o	o	o		Right great toe		Right great toe amputated
		E. III	Painful gangrene right third toe	o	o	o	Superficial and deep peroneal			Pain relieved. Toe healed
		E. IV	Painful gangrene right second toe	o	o	o	Superficial and deep peroneal, posterior tibial	Right second toe		Pain partially relieved. Toe healed
		E. V	Painful ulcer right third toe	o	o	o		Right third, fourth, and fifth toes		Right third toe amputated, fourth and fifth toes normal, amputated for prophylaxis
8. 300831	46	*Entry I*	Painful gangrene left great toe, ulcer fourth toe	+	o	+	Posterior tibial, superficial and deep peroneal	All toes, heads of metatarsals, second, third, and fourth		Great toe amputated; gangrene extended; second, third, and fourth toes amputated; finally fifth toe amputated because useless
		E. II	Painful gangrene right third toe	+	o	o	Posterior tibial and superficial peroneal	Right second and third toes		Pain immediately relieved. Infection spread; second and third toes amputated
9. 306379	29		Painful beginning gangrene right great toe	+	o	o	Superficial and deep peroneal, posterior tibial	Right great toe		Pain relieved but toe still sloughing. Toe amputated, healed well. All sensation returned after 2 mos.

TABLE I.*—THROMBO-ANGIITIS OBLITERANS—Continued

Case No. Hosp. No.	Age		Local lesion	Peripheral vessels Pop.	D.P.	P.T.	Nerves blocked	Minor amputation	Major amputation	End-result
10. 289606	43	*Entry I*	Painful ulcer with beginning gangrene in stump of right great toe and between fourth and fifth toes	o	o	o	Posterior tibial, superficial and deep peroneal	Right second and fifth toes		Pain relieved. Gangrenous areas became sharply defined; second and fifth toes amputated. Foot amputation advised but patient refused. Discharged with open areas improved but still some gangrene. All nerves regenerated 1 year later; 2 years later still one clean ulcer open
		E. II	Amputation stump of left fifth toe unhealed and painful	o	o	o	Superficial peroneal and posterior tibial	Left fourth toe		Left foot amputation advised but patient refused. Gangrene developed in fourth left toe, which was then amputated
11. 305749	35		Painful beginning gangrene great toe and lateral aspect right foot	o	o	o	Sural	Left fifth toe		Ulcer healing and painless on discharge
12. 312037	33	*E. I*	Pain in all toes of right foot, great and fifth toes infected	+	o	o	Posterior tibial and superficial peroneal			Toes healing, pain relieved for 2 months
		E. II	Pain in right great toe recurrent (with osteomyelitis)	+	o	o	Deep peroneal, saphenous, sural	Right great toe		Right great toe amputated and sole of foot drained. Healing on discharge
13. 307568	37		Left great toe amputated for gangrene, pain in stump	o	o	o	Posterior tibial, superficial peroneal		1. Left foot at ankle 2. Gritti-Stokes	Gangrene progressed in foot, amputated at ankle 15 days after injection, Gritti-Stokes 3 days later
14. 303113	44		Painful gangrene right third toe	+	+	o	Posterior tibial, superficial and deep peroneal		Right leg below knee	Pain completely relieved but recurred after 1 month. Reinjected. Gangrene progressed to ball of foot and pain recurred. Amputation below knee done 6 weeks after injection
15. 311648	33		Infection in left great toe, moderate pain in foot	+	+	+	Posterior tibial			Toe improved but still unhealed on discharge. Nerves all regenerated 6 months later
16. 297582	36		Painful sinus right fifth toe	+	o	o	Sural, posterior tibial, saphenous	Right fourth and fifth toes		Pain relieved. Fourth and fifth toes healed slowly and completely
17. 307647	39		Right second, third, and fourth toes amputated, stumps unhealed and sloughing, severe pain in foot, especially great toe	o	o	o	Posterior tibial, superficial and deep peroneal		Gritti-Stokes, right	Gangrene developed in great toe, incisions for nerve block did not heal, pain continued. Gritti-Stokes amputation done 2 weeks after injection
18. 320706	34		Amputation stumps of right great and fifth toes painful on dressing	o	o	o	Sural, superficial peroneal, posterior tibial	Right first and fifth metatarsals		Stumps of both toes broke down 2 months after injection, but only slightly painful. Head of first metatarsal removed, fifth metatarsal stump curetted. Four months later sequestrectomy of non-healing great toe stump. Took 5 months more to heal. Nerves regenerated 8 months after block
19. 319479	39		Painful ulcer right great toe, dry ulcer second toe	+	o	o	Superficial and deep peroneal, posterior tibial	Right great and second toes		Pain relieved but toes did not heal. Second toe amputated 9 days after alcohol injection. Great toe developed gangrene; 2 months later amputated
20. 310346	24		Painful gangrene in stump of first metatarsal right	o	o	o	Superficial and deep peroneal, posterior tibial, sural		1. Foot at ankle 2. Gritti-Stokes	Entire dorsum of foot became ulcerated and gangrenous, amputation at ankle, then Gritti-Stokes
21. 310922	35		Painful ulcer left great toe, whole foot discolored	+	o	o	Superficial and deep peroneal			Lesion healed and pain-free 3 months after nerve block

*The average duration of symptoms was 3 years, 6 months in the thrombo-angiitis obliterans group, and 1 year, 8 months in the arteriosclerosis group. The interval between serious involvement of the two legs in the cases in which bilateral nerve block was necessary was 2 years.

TABLE I.*—THROMBO-ANGIITIS OBLITERANS—Continued

Case No. Hosp. No.	Age		Local lesion	Peripheral vessels Pop.	D.P.	P.T.	Nerves blocked	Minor amputation	Major amputation	End-result
22. 309660	46		Painful ulcer right fifth toe	o	o	o	Sural			Patient returned to hospital 3 weeks after nerve block with ulcer unhealed, though pain improved. Nerve block incision unhealed. After 2 months toe ulcer healed, incision still open. Grafts applied, periarterial sympathectomy with excision and ligation of femoral vein done without effect on healing of nerve block incision. Lumbar sympathetic ganglionectomy done, followed by healing
23. 318377	48		Painful threatened gangrene of toes of left foot, ulcer at base of great toe	+	o	o	Posterior tibial, superficial peroneal, sural	Great and fifth toes left	Gritti-Stokes, left	Gangrene progressed, great and fifth toes amputated; gangrene appeared in fourth toe; Gritti-Stokes amputation of leg done. Pain completely relieved
24. 303537	39	*Entry I*	Painful gangrene right great toe, beginning gangrene second toe	+	o	o	Posterior tibial, superficial and deep peroneal	Right great toe		Dry gangrene at tip of great toe removed. Anæsthesia complete and ulcers healed 5 months after nerve block
		E. II	Ulcers of great and second toes, left	o	o	o	Superficial peroneal, posterior tibial	Left second toe		Ulcers did not heal. Gangrene developed on second toe. Toe amputated, followed by painless healing
25. 320283	26		Right great toe nail bed infected, whole foot discolored and painful	o	o	o	Superficial and deep peroneal, posterior tibial	Right great toe	Right lower leg	Gangrene developed in great toe. Toe amputated. Stump failed to heal. Re-amputated first metatarsal. Infection spread. Guillotine amputation of lower leg done, followed by re-amputation 5 inches below knee
26. 297089	38	*Entry I*	Left great toe nail bed sloughing	+	o	o		Left great toe		Circulation not improved by medical measures. Toe amputated. Amputation wound ulcerated. Lumbar ganglionectomy done, stump grafted, wound healed 4 months after amputation
		E. II	Right third and fourth toes ulcerated	+	o	o				Right lumbar ganglionectomy done. Healing not effected
		E. III	Right third and fourth toes ulcerated	+	o	o	Posterior tibial, superficial and deep peroneal	Right third and fourth toes		Right fourth toe amputated. Nerve block done for pain in stump. Then third toe amputated. Slow healing, complete in 6 months
		E. IV	Ulceration between left fourth and fifth toes	o	o	o	All 5 nerves	Left fourth and fifth toes and metatarsals		Pain relieved but healing not effected. Toes amputated. Slow healing complicated by osteomyelitis. Nerve blocks repeated on return of pain
		E. V	Ulceration left third toe	+	o	o		Left third toe		All remaining toes of both feet to be amputated
27. 313881	31		Painful ulcers tips of fourth and fifth toes	o	o	o	Sural, posterior tibial, superficial peroneal		Gritti-Stokes, left	Pain relieved temporarily, but 6 weeks later gangrene commenced in fifth toe with severe pain. Superficial peroneal nerve blocked again. Gangrene extended into foot, poor healing of nerve block incision. Gritti-Stokes amputation done
28. 314738	54		Pain and swelling left foot, ulcers on leg at sites of 2 previous nerve injections	o	o	o	Superficial and deep peroneal, sural, saphenous		Low thigh, guillotine, left	Gangrene developed, low thigh amputation done. Amputation stump painful a year later. Posterior femoral cutaneous nerve blocked. Pain not completely relieved. Stump re-amputated, neuromata found. Later cordotomy for pain

*The average duration of symptoms was 3 years, 6 months in the thrombo-angiitis obliterans group, and 1 year, 8 months in the arteriosclerosis group. The interval between serious involvement of the two legs in the cases in which bilateral nerve block was necessary was 2 years.

TABLE I—Continued—ARTERIOSCLEROSIS

Case No. Hosp. No.	Age	Local lesion	Peripheral vessels Pop.	D.P.	P.T.	Nerves blocked	Minor amputation	Major amputation	End-result
29. 284284	43	Beginning gangrene of first 3 toes, left	o	o	o	Cutaneous branch of peroneal, and posterior tibial		Gritti-Stokes, left	Gangrene and pain spread rapidly to lower leg. Gritti-Stokes amputation
30. 296230	70	Threatened gangrene of right foot, severe pain in first 3 toes	o	o	o	Cutaneous branch of peroneal, deep peroneal, and posterior tibial		Gritti-Stokes, right	Painless gangrene of foot in 10 days. Gritti-Stokes amputation
31. 293698	70	Non-healing ulcer lateral lower third of leg	+	o	o	Peroneal anastomotic and external saphenous branches of sural nerve			Ulcer healed slowly and painlessly. Incision also healed slowly, with minor sepsis
32. 298162	67	Ulcer of fifth toe with septic joint. Continued pain after amputation of toe	+	o	o	Posterior tibial, cutaneous peroneal, and musculo-cutaneous	Left fifth toe		Has had occasional ulcerations on foot since. Still has useful foot and pain not severe enough to warrant risk of amputation
33. 297657	67	Painful gangrene of left great toe	o	o	o	Posterior tibial		Lower thigh, left	Relief of pain. Gangrene advanced slowly, forcing amputation of leg
34. 298499	62	Painful gangrene of right fifth toe	o	o	o	Posterior tibial, cutaneous peroneal		Gritti-Stokes, right	Rapid spread of gangrene. Amputation of leg done
35. 318029	55	Large painful ulcer over right tibia, pain extending into foot and up to groin	o	o	o	Saphenous		Right lower thigh	Ulcer continued dirty and sloughing. Leg amputated at mid thigh 10 days after injection
36. 309942	56	*E. I* Painful ulcer side of right foot	o	o	o	Sural			Ulcer healed but pain not relieved
		E. II Ulcer side of right foot healed but still painful	o	o	o	Sural			Pain completely relieved for 3 mos.
		E. III Painful ulcer recurrent	o	o	o	Superficial peroneal			Ulcer practically healed, no pain, no regeneration after 2 months
37. 309599	72	Painful ulcer of left fifth toe	o	o	o	Sural, superficial peroneal, posterior tibial	Left fifth and fourth toes		Ulcer improved, but toe amputated through part of proximal phalanx; 2 months later clean ulcer over stump; 8 months later ulcer healed. Fourth toe gangrenous; this amputated. Nerves still completely blocked
38. 318084	60	Painful gangrenous stump of right fifth toe	o	o	o	Sural, posterior tibial, superficial peroneal	Right fifth metatarsal	Right leg	Amputation wound of fifth toe did not heal. Leg amputation done 3 months later
39. 314173	70	Painful ulcer with beginning gangrene in left fifth toe	+	o	o	Sural, posterior tibial, superficial peroneal			Slow healing. Pain relieved
40. 321022	56	Gangrene of left great toe with severe pain	o	o	o	Posterior tibial, superficial and deep peroneal			Wound healing, pain gone except for "burning" on dorsum of foot on discharge
41. 322083	61	Painful ulcer on dorsum of left foot	+	o	o	Superficial peroneal		Gritti-Stokes, left	Ulcer failed to heal, lymphangitis developed, Gritti-Stokes amputation
42. 317949	41	Amputation stumps fourth and fifth toes unable to tolerate dressings	o	o	o	Posterior tibial, sural, superficial peroneal			Stumps unhealed on discharge. Amputation of leg advised, refused by patient. Pain entirely relieved.
43. 314650	71	Painful ulcer sole of left foot with discoloration of whole foot	o	o	o	Posterior tibial, sural, superficial and deep peroneal		Left lower leg	Patient returned to hospital 4 weeks after last nerve block with ulcer unhealed and painful, lymphangitis, and threatened gangrene. Amputation at lower third of femur
44. 313181	82	Continued pain in stump of right second toe after amputation for gangrene	o	o	o	Superficial and deep peroneal, posterior tibial		Low thigh right	Gangrene spread into foot. Low thigh amputation done
45. 309406	69	Painful beginning gangrene of right foot, especially third and fourth toes	+	o	o	Superficial peroneal		Gritti-Stokes, right	Gangrene spread and became more painful. Gritti-Stokes amputation done 4 months after block

Fig. 2. Method of injecting alcohol into a nerve. Alcohol is injected into a small segment of nerve defined by two snug ties. These are later removed. We feel this prevents irregular diffusion of the solution along the nerve, which may result in paræsthesia rather than anæsthesia. The duration of anæsthesia depends upon the length of the segment of nerve which is injected.

should be followed by lumbar ganglionectomy in selected cases. Lastly, the anæsthetic area is dry. This diminishes heat loss from the skin surface by radiation and convection, which are much greater in the presence of moisture.

RESULTS

It is of course difficult to compare the results of this series with those obtained in a similar series not treated by this method. Table II gives a comparison between the first 25 consecutive cases of thrombo-angiitis obliterans treated by this method and the preceding 25 similar cases treated by other methods. It shows a marked increase in the number of successful minor amputations and a marked decrease in the number of major amputations necessary in the cases treated by nerve block. We would particularly call attention to the decrease in the number of necessary bilateral major amputations in the nerve block series. Table III shows the distributions of major and minor amputations in this series of 45 cases. Again we would emphasize the fact that only one bilateral major amputation was necessary in this series. Five cases required amputation of one or more toes in both feet, but were saved from bilateral major amputations. In our opinion these good results were due solely to this operation. Figures 4 and 5 show end-results in 2 cases.

TABLE II.—THROMBO-ANGIITIS OBLITERANS

	25 cases without nerve block	25 cases with nerve block
Amputation unnecessary	1	4
Minor amputations	7	14
Unilateral major amputations	11	6
Bilateral major amputations	6	1
Percentage of involved extremities having major amputations	74.2	30.8

Showing a decrease in the number of cases in which no amputation is necessary, an increase in the number of successful minor amputations, a decrease in unilateral major amputations, and a striking decrease in the number of bilateral major amputations in the nerve block cases.

The results are better in the patients with thrombo-angiitis obliterans than in the arteriosclerotic group, as shown in Table IV, where the influence of the different pathological processes is brought out. As has been previously emphasized, the average age is much less and the duration of symptoms much longer in the patients suffering with thrombo-angiitis obliterans (1.) This, of course, gives a much better opportunity for the development of collateral circulation. The presence or absence of popliteal pulsation is important in both groups from the point of view of end-results, but less so in the case of thrombo-angiitis obliterans. Here one is much more likely to avoid a major amputation in the absence of popliteal pulsation than in the arteriosclerotic group (Table V).

To speak for a minute about minor amputations: We are impressed by the fact that the difficulty usually involves one or two toes, rarely more. After these have been successfully healed with the aid of minor amputations, there are left two or three useless toes which in our experience are very apt to get the patient into future serious difficulty because of minor trauma and poor hygiene. In several cases we have had to block the same nerves several times on different entries for successive gangrene of one toe after another. The nerves had regenerated between admissions. We are favorably impressed by the results of prophylactic amputation of these remaining superfluous toes by the method of lateral flaps through the middle of the proximal phalanx, as

TABLE III.—AMPUTATIONS

	Disease		
	Thrombo-angiitis obliterans	Arteriosclerosis	Total
Bilateral major	1	0	1
Unilateral major	10	10	20
Bilateral minor	5	0	5
Unilateral minor	8	3	11
Amp. unnecessary	4	4	8
Total	28	17	45

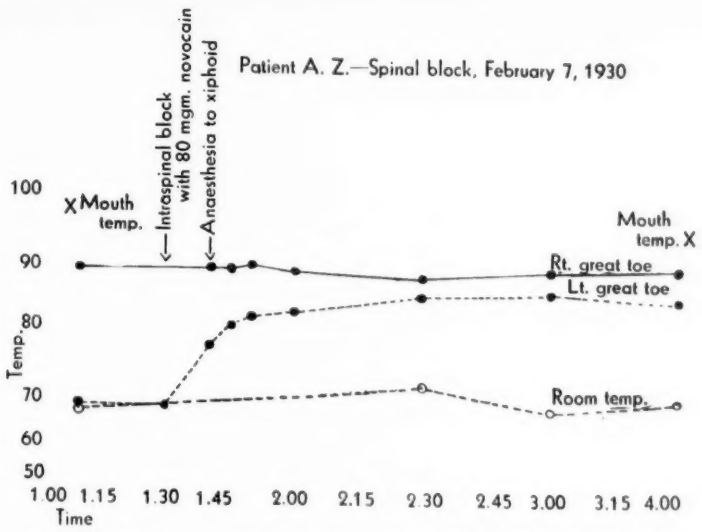

Fig. 3. Surface temperature rise after nerve block. Case 24 (Table I). Chart showing a surface temperature rise of nearly 20 degrees F. in the right great toe following nerve block. After spinal anæsthesia, which paralyzes the lumbar sympathetic trunks, there is no further rise of surface temperature in this toe, suggesting that it has been very completely sympathectomized by peripheral nerve block. The surface temperature of the left great toe, however, does rise approximately 18 degrees F. for the duration of the spinal anæsthesia. (Reproduced by permission, *New England Journal of Medicine*, 3.)

shown in Figure 5. This almost ensures the patient against future episodes of ulceration and gangrene, which usually start in the tip of a toe and rarely in the foot itself. The resulting foot with the heads of the metatarsals preserved is functionally satisfactory, provided the toe of the shoe is stuffed with sponge rubber or lamb's wool to prevent sliding forward into the shoe. At times a ¼ inch metatarsal bar is a great help.

Practically all of the major amputations in this series were of the low thigh or Gritti-Stokes type. We are not favorably impressed by the end-results in lower leg (point of election) amputations for advanced obliterative vascular disease. Recurrent ulceration and pain are so frequent that they become of great economic importance, and in many cases prevent the patient from using his permanent leg much more than 50 per cent of the time. The only cases in which we occasionally do point of election amputations are young individuals who are thin, and in whom the probability of loss of the other extremity is great. We have several patients (only one in this series) who have had bilateral Gritti-Stokes amputations and get about very well on a pair of short legs. From the preceding remarks it is obvious that major amputations are still necessary at times, although we

TABLE IV

Disease	Extremities involved	Major amputations number	per cent
Thrombo-angiitis obliterans	34	12	35.3
Arteriosclerosis	17	10	58.8

Showing the influence of the pathological process upon the loss of extremities. Major amputations are relatively more frequent in the arteriosclerotic group.

TABLE V

Disease	Number of extremities	Major amputations Number	Per cent
Popliteal pulsation present—			
Thrombo-angiitis obliterans	16	4	25.0
Arteriosclerosis	7	2	28.5
Popliteal pulsation absent—			
Thrombo-angiitis obliterans	17	8	47.1
Arteriosclerosis	11	8	72.7

Showing the influence of extent of obliteration upon the end-result. Major amputations are more frequent in the absence of popliteal pulsation, particularly in the arteriosclerotic group.

Fig. 4. End-result, Case 3 (Table I). Loss of second and third toes of right foot, and all toes and heads of all metatarsal bones of left foot.

Fig. 5. End-result, Case 26 (Table I). After multiple entries, minor amputations, and repeated nerve block, the first, third, and fifth toes of the right foot and the third toe of the left foot were amputated before they became involved.

feel that this operation has enabled us to more than double the number of successful minor amputations and to more than halve the number of major amputations. We believe it has also increased the number of cases in which no amputation is necessary. We still intend to do major amputations only in the presence of hopeless gangrene or sepsis which endangers the life of the patient.

CONCLUSIONS

1. Peripheral nerve block is an effective method of controlling rest pain in patients with advanced obliterative vascular disease.

2. Besides controlling pain, the circulation to the part may be increased because the anæsthetic area is also deprived of its vasoconstrictor nerves.

3. Local infection in open lesions is more easily controlled because frequent antiseptic dressings can be done painlessly.

4. The general condition of the patient improves with the relief of pain, and other conservative measures become more effective.

5. The operation requires careful technique, asepsis, and should be done in multiple stages. Under these circumstances complications are few.

6. The nerves may either be injected with alcohol or crushed. The latter method is more simple and equally effective, although the nerves regenerate more rapidly.

7. We believe that this procedure has more than halved the number of necessary major amputations, and more than doubled the number of successful minor amputations. It also has increased the number of cases in which no amputation is necessary. We would particularly emphasize the striking reduction in bilateral major amputations in this series.

8. The results are better in the patients with thrombo-angiitis obliterans than in the arteriosclerotic group. Patients with pulsation of the popliteal vessels do better than those in which the vessel is obliterated at this level.

9. Amputation of uninvolved toes is an effective prophylactic procedure in selected cases. If the vasomotor index is high, lumbar ganglionectomy should be done after the peripheral nerves have regenerated in certain cases.

10. Unless hopeless gangrene or infection sufficient to endanger the life of the patient is present, this operation should be tried before resorting to a major amputation. It is not indicated in the early treatment of obliterative vascular disease, but should be used in advanced stages after other conservative methods have proved inadequate.

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URETERO-INTESTINAL IMPLANTATION WITH DRAINAGE BY EXTRAPERITONEAL CATHETER

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IN 1925, Smitten reviewed 755 cases of ureterointestinal implantation reported in the literature with an average surgical mortality of 33.6 per cent. Twenty-nine per cent of the patients died of peritonitis and 23.4 per cent of severe pyelonephritis. Shock and pneumonia were common complications. The course of patients surviving the operation was not given. Doubtless many of them died of ascending infection or from slow renal obstruction.

This is a good representation of the experience of others. To put the ureters in the bowel always involves the two risks of infection and obstruction. The facts that birds and some reptiles normally have ureters ending in a cloaca full of pathogenic organisms, and that many surgeons have been successful in imitating this anatomical relationship to the extent that patients live many years with neither of the two complications, account for the belief of many surgeons that a method which will minimize these great risks will be found. Surgical efforts thus inspired go in waves, and there is one of limited enthusiasm sweeping the country at present.

Almost every conceivable idea to prevent infection and ureteral obstruction has been tried. One of the important causes of failure with many methods has been the obstruction to a free outflow of urine caused by the congestion and œdema at the site of implantation. Temporary obstruction from this cause may be the starting point for an acute obstructive pyelonephritis with multiple abscesses. If only one ureter has been transplanted, this renal infection may subside and the kidney recover when urine begins to drain freely following the disappearance of the local œdema. If bilateral transplantation has been done, the condition is much more serious. Statistics clearly show that, in case of exstrophy, it is safer to transplant one ureter at a time. In old people with cancer of the bladder, however, every extra stage of surgery multiplies the risk by that much and a technique is needed which does not increase the risk if both ureters are transplanted at the same operation. Higgins's modification of Coffey's technique III[1] and Poth's modification of Higgins's technique[2] are good for two reasons, and have been performed successfully. In the first place, the bowel is not opened and the technique is aseptic. In the second place, urine drains naturally into the bladder and the intestinal wound about the ureter heals without contamination either of urine or fæces and without producing urinary obstruction caused by the œdema of surgical trauma. However, drawing the ureter over to the rectum for anastomosis without dividing it may be difficult sometimes and if the ureter is on tension when the operation is done, the ureter may be found detached at the second operation and the whole purpose be defeated.

[1] First stage: The ureters are exposed but not divided. A portion of each is imbedded in a suitable channel of the muscular layer incised to the submucosa. The hidden intestino-ureteral stitch (lumen to lumen) is placed. Urine drains naturally until the hidden stitch cuts through. Second stage: Each ureter is divided just below the portion which has been imbedded.

[2] First stage: The ureters are imbedded as above but the hidden stitch is not used. The lumen of the ureter is not connected with the lumen of the bowel at this time. Second stage: An opening of the ureter into the bowel is made with a cautery passed up the ureter after its division below the portion imbedded and this cautery is drawn on out by an assistant through a proctoscope in the rectum.

Fig. 1. The right ureter is dilated and straight and the pyelogram shows cavity formation, indicating tuberculosis. The urine from the right kidney shows tubercle bacilli. There is questionable involvement of the upper pole on the left. The urine from the left kidney is negative.

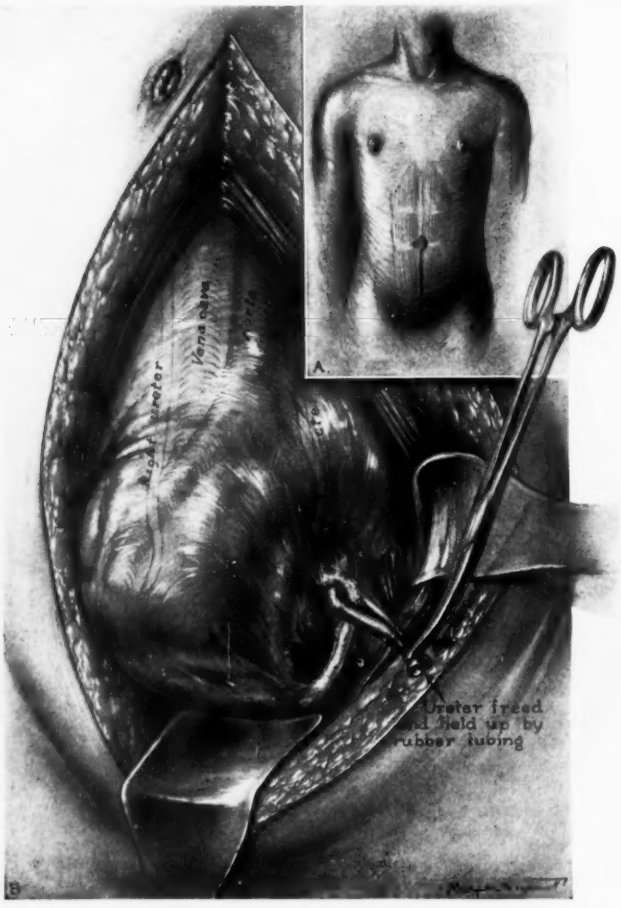

Fig. 2. A, The incision. B, Extraperitoneal exposure of the ureter which is ligated and divided near the bladder after the peritoneum has been opened and the abdomen inspected, as shown in Figure 3, and not at this stage.

A well known method of diversion of urine during the period of healing is nephrostomy. Preliminary drainage by nephrostomy reduces the risk of implantation but adds another operation. Of 7 patients on whom this was done 6 survived. The 1 who died (2 days after bilateral implantation, by Coffey's catheter technique, of shock and acute dilatation of the stomach) had an extensive carcinoma of the bladder and urethra. Unilateral implantation was done in each of the 6 patients who recovered. Each had one kidney and a contracted tuberculous bladder, nephrectomy for tuberculosis having been done sometime previously in all. Two are still living comfortably; 1 lived for 3 months and the others for over a year. Four of the 6 patients died of non-surgical causes long after operation and, except in 1 patient, the cause of death was unrelated to the implantation. One died in 3 months of miliary tuberculosis (autopsy), 1, in 13 months of right-sided lobar pneumonia (autopsy), 1, in 3 years of unknown cause (no autopsy), probably renal insufficiency, and 1, in 4½ years of calculous anuria (stone in the implanted ureter) (autopsy). Nephrostomy was indicated in these cases of tuberculosis as a therapeutic measure in itself

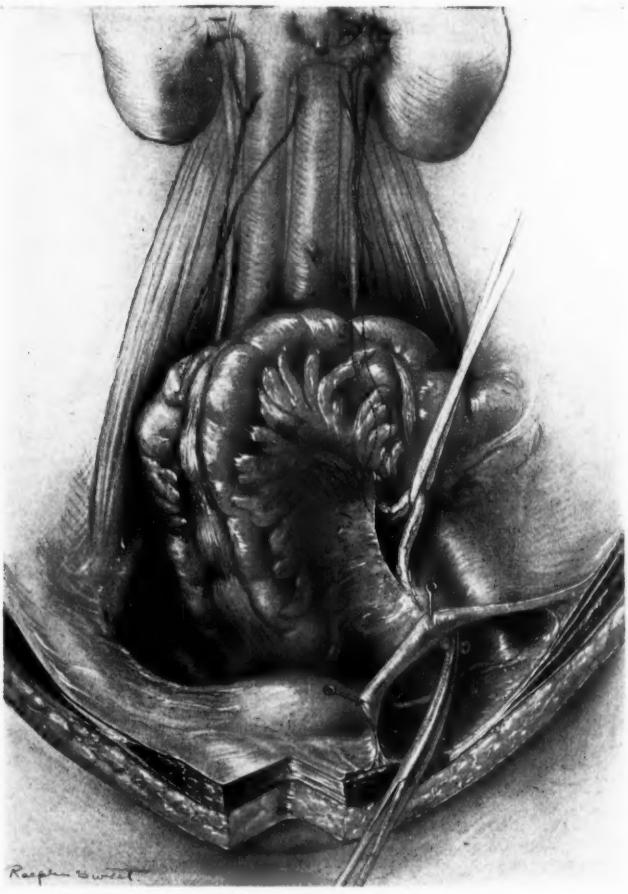

Fig. 3. The peritoneum has been incised alongside that portion of the pelvic colon favorable for ureteral anastomosis and the ureter, after division near the bladder, is drawn out through this incision.

regardless of its relationship to ureteral implantation. Finding that the remaining kidney is free of tuberculosis, demonstration of a fairly normal ureter and of restoration of function, when it had been reduced before nephrostomy, give the indications for implantation of the ureter. Patients with contracted, hopelessly involved bladders get along quite comfortably with simple nephrostomy. Three such patients are now living 5, 2, and 1 years, respectively, with symptomless bladders and nephrostomy drainage, one because he prefers the nephrostomy tube to another operation and the other two because the lower ureters are so badly diseased that the operation never has been proposed. However, nephrostomy should not be the routine procedure in this type of case. The soundness of the remaining kidney and ureter can be determined by tests of total function and excretory urography even when the ureter cannot be catheterized, and the ureter can be implanted at once without preliminary nephrostomy. Recently, 2 patients, each with a nephrectomy for tuberculosis, and a tuberculous bladder but with good renal function and a normal appearing ureter, have weathered direct implantation of this solitary ureter by a simple seven suture method without drainage (to be reported). The other tuberculous patients in poor

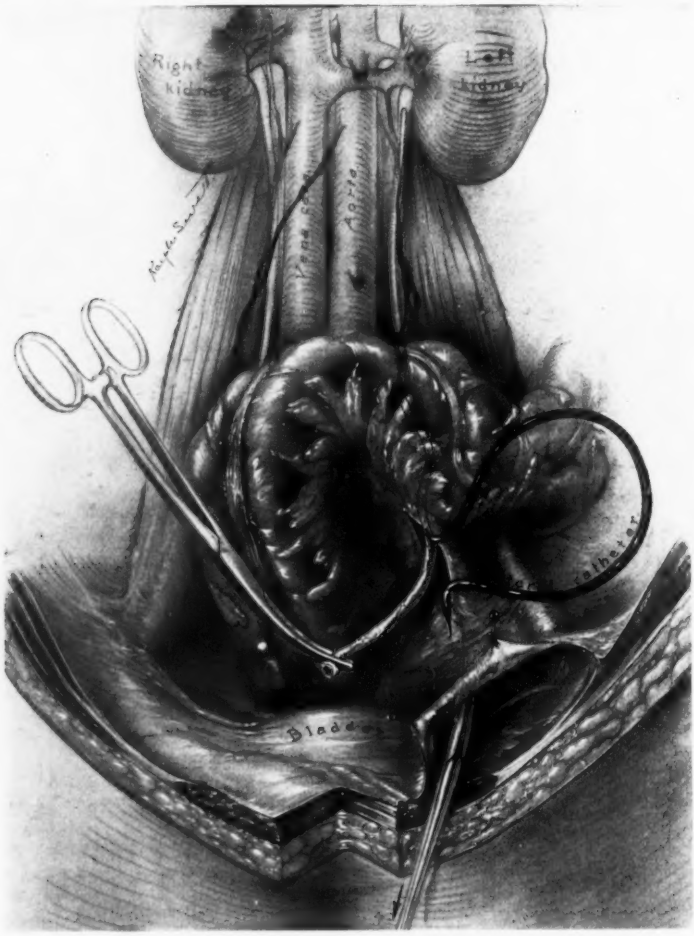

Fig. 4. The ureter is incised as high as possible under the peritoneal flap and a retention catheter is inserted. The end of this is brought out retroperitoneally.

condition succumbed from nephrostomy alone. Drainage by preliminary nephrostomy should not be regarded as a stage of uretero-intestinal implantation. It only complicates the problem by adding more surgery. It has, however, its own indications. In some patients wearing a nephrostomy tube, uretero-intestinal implantation may become indicated and the fact that good drainage already is established lowers the risk of operation.

Coffey's enthusiasm over the results of catheter drainage (technique II) as a preventive of obstruction, thus permitting bilateral implantation at one operation, has led to its trial by a great many surgeons. In memory of the debt owed this surgeon for his faithful prolonged study of the problem, it should be said that he himself regarded this method as imperfect and, at the time of his death, was working for a better one. The catheter, which is tied to the ureter and therefore cannot be withdrawn until the ureter sloughs off, is in itself a cause of complications, some of which may be fatal. Trouble originating directly from the catheter occurred in 6 of my 15 patients operated upon by this technique and 2 died in consequence of such complication. Table I.

Extraperitoneal drainage after ureterotomy for stone at any portion of the ureter is a common experience of urologists. When Coffey first dis-

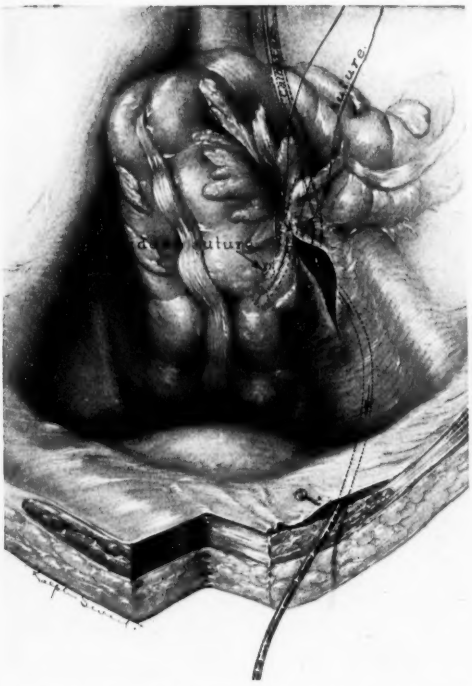

Fig. 5. The ureter has been implanted by technique III of Coffey and the site of implantation is covered by the flap of peritoneum, leaving the retention catheter to drain extraperitoneally.

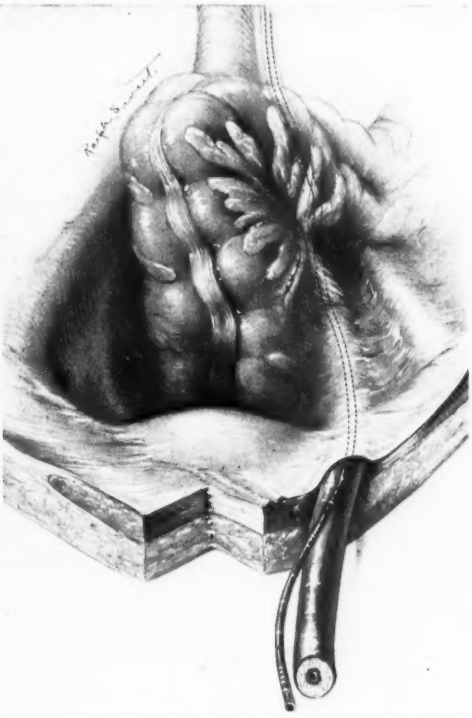

Fig. 6. Illustrates how the area of implantation can be drained retroperitoneally.

cussed the aseptic advantages of the hidden suture (technique III) it occurred to me that extraperitoneal drainage, as after ureterolithotomy, might be used to advantage in conjunction with this technique. The trial of this method has been made in 3 patients, with a good result in 2, but a fatal complication from the catheter in the third. The cases are reported in order to place on record this modification of method and at the same time to condemn it. A retention catheter in the ureter draining extraperitoneally carries the same element of danger as Coffey's Technique II with catheters draining transrectally. The accompanying illustrations explain the method as well as would description in detail.

CASE REPORTS

CASE 1. Mrs. C., aged 41 years, complained of frequency and nocturia for 7 months (June, 1932).

A cystoscopic examination on December 28, 1932, showed the bladder to be small and contracted with a capacity of 100 cubic centimeters. There was œdema about the ureteral orifices. The wall of the bladder was reddened. The right kidney was loaded with pus which was positive for tuberculosis. The left kidney showed 5 to 10 white blood cells per high dry field. The acid fast test was negative. The phthalein test showed some diminution of function on the right side. The total function was 43.5 per cent in one-half hour. Bilateral pyelograms revealed caseocavernous tuberculosis of the right kidney and were suggestive of some involvement at the upper pole of the left kidney (Fig. 1).

A right nephrectomy was done on February 1, 1933, and the pathological report showed tuberculosis of the kidney.

The patient's postoperative course was uneventful except for a small hæmatoma in the posterior angle of the wound which was drained adequately. At the time of discharge, there was no elevation of temperature. There was a small sinus of the posterior angle of the wound, draining very little. The phthalein test showed, first hour, 2 per cent; second hour, 30 per cent; total, 32 per cent.

The patient was readmitted on October 19, 1933, complaining of marked urgency and frequency so that she had to sleep on a bedpan. There was constant severe pain in the lower abdomen. The symptoms had been worse since nephrectomy. The urine was cloudy. There had been no loss of weight and no respiratory symptoms.

The blood count showed red blood cells, 5.1 million; hæmoglobin, 95 per cent; blood pressure, 155/80. The urine was cloudy, without albumin or sugar, but was loaded with white blood cells. Phenolsulphonphthalein test on October 20, 1933, showed first hour, 20 per cent; second hour, 15 per cent; total, 35 per cent.

Fig. 7. Excretory urogram (5 minutes) of the first patient a year after uretero-intestinal implantation.

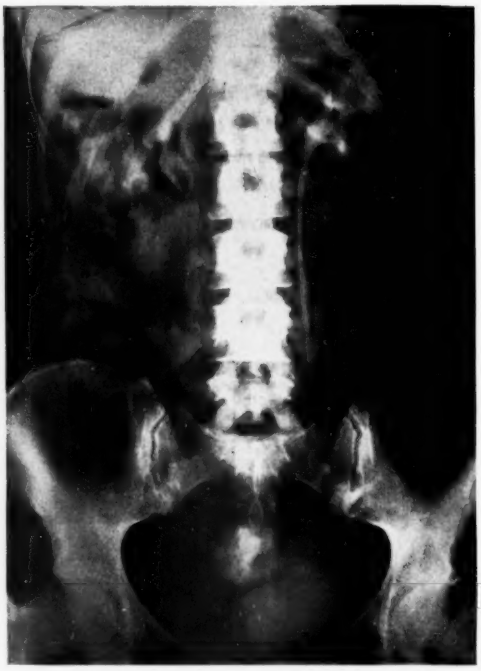

Fig. 8. Excretory urogram of the second patient 2 weeks after operation. The left kidney and ureter show normal function and appearance; the right, poor function and dilatation. The rectum and pelvic colon are outlined.

On October 21, 1933, transplantation of the left ureter into the rectum was done under spinal anæsthesia with gas and oxygen. The patient's blood pressure was 60/38 at the beginning of the operation after the spinal anæsthesia and 104/54 at the end of the operation. She left the table in fair condition. Her systolic blood pressure was 70, one-half hour after operation. The patient responded to glucose administered intravenously.

Operative procedure: An abdominal incision was made slightly to the left of the midline. The ureter was exposed (Fig. 2) by dissecting up the peritoneum above the brim of the pelvis. The peritoneum then was opened in line with the abdominal incision. The intestines were packed off. The peritoneum was slit as near the pelvic colon as possible and the ureter drawn through this opening (Fig. 3). A small longitudinal slit was made in the ureter high in the area of the peritoneal flap (Fig. 4). A No. 10 catheter was put through into the ureter. The distal end of this catheter was brought out through the abdominal wound behind the peritoneum (Fig. 5). Urine was seen to trickle out from the catheter. The ureter was implanted at the selected area in the bowel, Coffey's technique III being used. Drains were placed retroperitoneally (Fig. 6) and the wound was closed with retention sutures of wire.

The catheter drained well. On October 23, a catheterized specimen of urine showed 75 white blood cells per high dry field, 7 red blood cells per high dry field, and a few rods. The urine was alkaline. The ureteral catheter was removed on November 1; the urine was all draining through the wound. The stool was soft and formed. The patient began passing urine by rectum 18 days after the implantation, and, within 6 days, the urine was passed entirely by rectum.

The patient was discharged on November 23, 1933, afebrile, and with all the urine being passed by rectum; the bowel movements were regular. The wound was well healed except for some infection at the lower end. The pain in the bladder was relieved. She has been seen several times and remains well and comfortable (1 year). Figure 7 shows the last excretory urogram.

Case 2. Mrs. L. B., aged 32 years, entered the hospital on October 25, 1933. There had been three previous entries, two in May and a third in June, 1933. At the time of the first admission, a diagnosis of epidermoid carcinoma of the vagina had been made and the subsequent entries were for the purpose of X-ray and radium therapy. This consisted of approximately 3000 millicurie hours of radium and 10 X-ray treatments. Her progress had been followed closely in the Women's Clinic.

Since July, 1933, she had had a foul vaginal discharge. Part of the severe lower abdominal pain had been relieved by X-ray therapy but had recurred with increased intensity several weeks before the present admission. The pain was continuous, was made worse by exertion, and seemed most severe at night. It involved the right lower quadrant of the abdomen, the back and the right hip. The patient was still able to be up and to do part of her housework. Moderate bleeding from the vagina over a period of 3 to 4 hours occurred on October 13. She developed urinary frequency and nocturia four or five times.

The *physical examination* was essentially negative except for the local condition. The abdomen showed normal contour. The panniculus was fairly fixed. There was an old, well healed suprapubic scar. No masses were palpable. There was considerable tenderness over the entire right

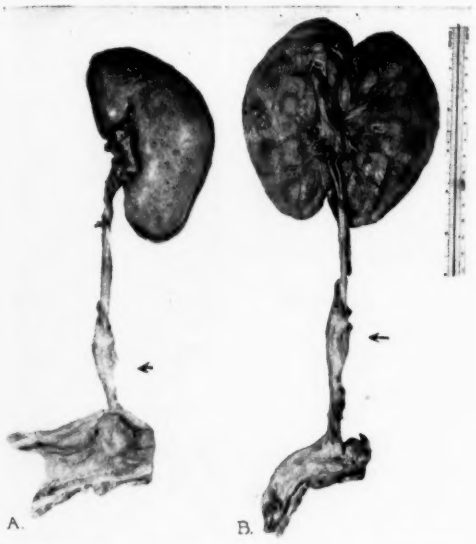

Fig. 9. The left ureter and kidney of the second patient. A, Shows the ureteral opening in the bowel. B, The kidney is neither infected nor its pelvis dilated. The entrance into the intestine appears to be direct and not oblique.

side of the abdomen. The liver edge was palpable at the costal margin. There was tenderness to pressure over the right sacro-iliac joint. On pelvic examination the vaginal vault was found to be contracted. The cervix was small. The area of the right vaginal wall was puckered and relatively well healed. Because of this fixation, nothing else could be felt beyond it. On rectal examination to the right, the parametrial region was found to be filled with a fan-shaped mass which extended outward and backward to the wall of the pelvis. Pressure on this region caused pain. The rectal lumen was not appreciably encroached upon. The blood pressure was 110/70.

The laboratory findings were as follows: blood count: red blood cells, 4,100,000; white blood cells, 4,800; differential: polymorphonuclears, 68 per cent; small lymphocytes, 20 per cent; large lymphocytes, 2 per cent; eosinophils, 4 per cent; mononuclears, 6 per cent; blood Wassermann test, negative; urine, negative. X-ray examination of the pelvis on October 28 showed no evidence of metastases to the bone.

On January 2, a laparotomy was done by Dr. Lynch for the purpose of exploration, the release of adhesions, and a right salpingectomy. Palpation of the parametrial region revealed that on the right side, a mass extended outward from the vaginal vault toward the sides of the pelvis. This did not seem quite to reach the pelvic wall. It was firm and very slightly movable. The left parametrial connective tissue was soft with no evidence of growth. No glands could be palpated in the pelvis. Because of the patient's youth, it was felt that it would be wise to attempt to remove the entire uterus, upper vagina, and bladder. This of course could not be done without preliminary transplantation of the ureters. The writer was called in to complete this part of the operation and a bilateral ureteral transplant to the sigmoid was done, the Coffey technique III being used, supplemented by bilateral ureterostomy with extraperitoneal drainage by catheter as in Case 1.

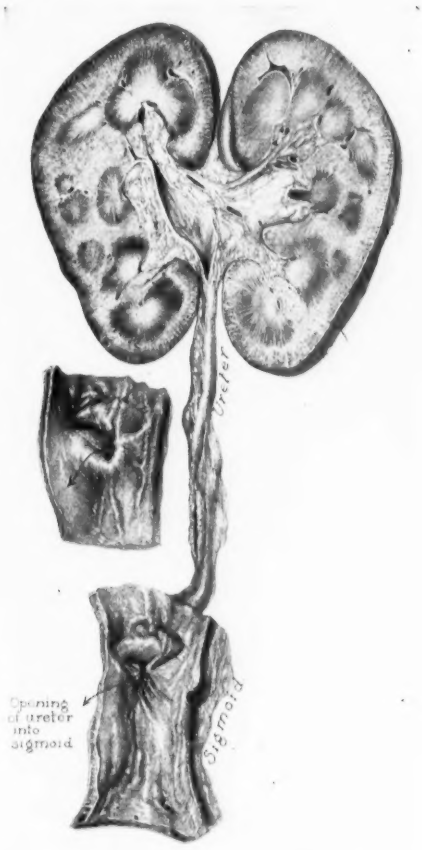

Fig. 10. Drawing of the successful implantation on the left side in Case 2 of 8 months' duration.

The patient's postoperative course was rather stormy. She developed a fæcal fistula through the wound and then an infection of the wound. Her temperature continued to rise to 38 and 39 degrees C. for many days.

On January 12, excretory urograms showed a normally functioning left kidney but hydronephrosis and hydroureter with a poor function for the right kidney. (Fig. 8.)

On January 13, a right nephrectomy was done. Considerable pus was found in the retroperitoneal area immediately surrounding the kidney and down the course of the ureter. The kidney appeared to be large. When opened, the pelvis was filled with pus. Microscopically multiple cortical abscesses of the kidney with chronic pyelitis and ureteritis were found.

After irrigation and dakinization of the wound, the patient's condition rapidly improved and the temperature returned to normal. The kidney incision was almost healed at the time of discharge; however, thick, grumous, foul smelling material continued to ooze from the midline

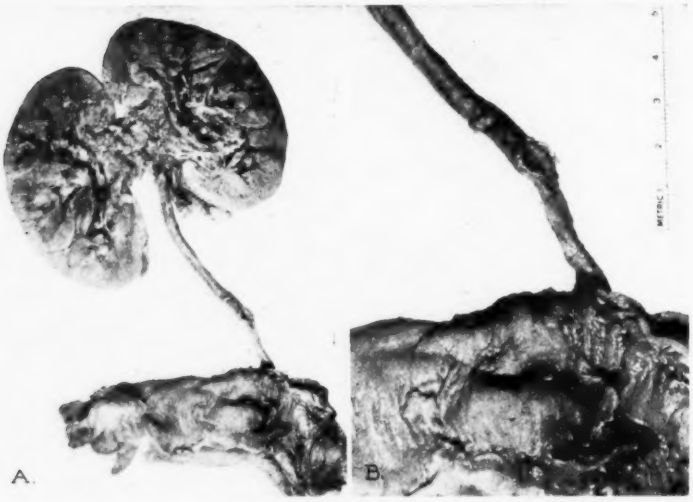

Fig. 11. Photographs showing the kidney, ureter, and a portion of the pelvic colon removed at autopsy in the third patient. A, The badly infected kidney and the marked ureteritis above the point at which the retention catheter was inserted. B, A closer view of the ureter.

suprapubic incision. The fistulous tract was extremely small. Pelvic examination on February 6 showed the vaginal mass to have increased markedly in size and to occupy practically the entire right lateral vaginal wall. Rectally it extended backward and to the lateral wall of the pelvis. Pressure upon it produced marked pain. The growth was obviously beyond any possibility of further surgery. Pain from the right pelvic region required repeated doses of opiates for relief.

At the time of discharge on February 16, the patient's appetite had returned, she had begun to gain weight and she was strong enough to sit in a chair for a short time each day.

After leaving the hospital she was comfortable except for pain in the right lower quadrant requiring for relief from 1 to 1½ grains of codeine two or three times daily. She re-entered the hospital on July 31, 1934, because of four severe hæmorrhages from the vagina. These recurred in spite of vaginal packs, and the patient died on August 10.

Autopsy showed numerous fine adhesions between the omentum and the abdominal scar. The omentum was adherent in the pelvis. Omental fat was thin and yellow. There was no free fluid. There were numerous adhesions in the right kidney fossa and about the cæcum. There was a small stump of appendix, 1 centimeter long, covered by adhesions, through which the cæcum was pulled medialward toward the base of the mesentery of the ileum.

Kidneys: The right kidney was absent. The left kidney was freely movable and surrounded by a thin layer of perirenal fat. It weighed 180 grams and was somewhat pale but possessed normal markings. The cortex was 0.7 centimeter thick and the pyramids were normal. The pelvis was about normal in size but the wall was fibrotic and thickened to 0.2 centimeter. The ureter was anastomosed to the sigmoid in the pelvis, an end-to-side anastomosis, which was patent through a collapsible opening which easily admitted a 3 millimeter probe. The ureter entered the sigmoid at about a right angle and the union was very firm (Fig. 9A and B). There was no excess ureter extending into the bowel. The ureter was somewhat dilated, just above the anastomosis, measuring 0.6 centimeter in diameter for about one-third of the distance, above which it was 0.4 centimeter in diameter. The wall was slightly thickened, uniformly. At the pelvic brim the ureter was adherent to the tumor mass but was not visibly constricted nor invaded (Fig. 10).

There was a hard nodular mass involving the posterior two-thirds of the vagina. This was most extensive on the ventral wall. It was thick, fixed, had a necrotic ulcerated surface, and discharged a foul serosanguineous exudate. The cervix was entirely eroded or replaced by the tumor. The neck of the uterus was likewise eroded and replaced, tumor tissue extending well into the body of the uterus, the lumen of which was obliterated by the growth. The tumor mass had invaded the bladder wall extensively on the vaginal and uterine surfaces.

Case 3. Mrs. Y., aged 68 years, had her first attack of hæmaturia 5 years before entry. The attacks recurred with increasing frequency, becoming severe 3 to 4 months ago. A large tumor of the bladder was found at cystoscopic examination 2 months ago. This was fulgurated three separate times without much regression. On January 6, 1934, the right ureter was transplanted to the sigmoid, with a ureterostomy drain, above the implant, which came out through the midline incision extraperitoneally. The bladder was removed. The patient was in poor condition in the surgery and the left ureter, which had been partially obstructed by the tumor in the bladder, was brought to the skin at the end of the operation. The patient excreted not more than about 700 cubic centimeters of urine each 24 hours. She developed tympanites and paralytic ileus with fæcal vomiting. A nasal tube was used for 3 or 4 days, with constant suction, fæcal material being aspirated. The patient failed rapidly during the last 5 days, developing uræmia and terminal bronchopneumonia. Non-protein

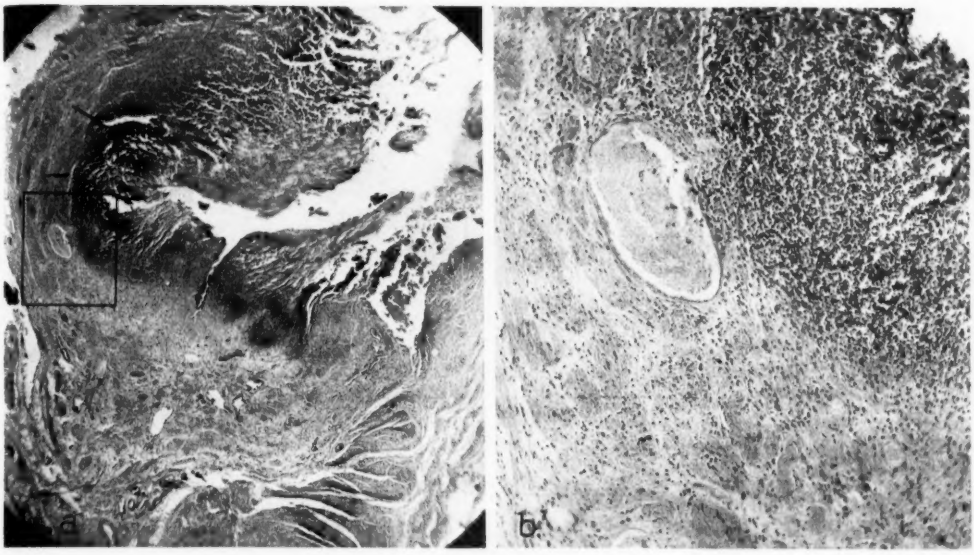

Fig. 12. a, Photomicrograph of a serial section showing the right ureter entering the sigmoid in Case 3. b, Higher powered photomicrograph showing the inflammatory reaction. The ureter shows atrophy and necrosis; at the point of entrance through the sigmoid, a subacute inflammatory reaction is seen extending in all the coats of the bowel.

nitrogen was 62.5, 2 days before death. Death occurred on January 19, 1934.

The autopsy (by Dr. Biskind) was limited to a slight lengthening of the midline abdominal incision, which showed very little healing and much induration as the result of an inflammatory reaction. The bowel showed a few loops plastered to the incision, forming a small pocket holding an inflammatory exudate. The pelvis was greatly distorted because of the absence of the bladder and, in this region, there were several hundred cubic centimeters of a thick purulent material which had collected in a pocket.

The gastro-intestinal tract was examined from the œsophagus to the rectum, and aside from the local reaction around the sigmoidostomy there was little evidence of abnormality.

The left kidney weighed 260 grams and was surrounded by a firm, perirenal fat which showed evidences of slight inflammation. The left ureter was greatly dilated and bluish red in color. The surrounding peri-ureteral tissue likewise was involved by inflammation. This ureter led to the stab wound in the left lower quadrant, and passed along the brim of the pelvis near the end of the left broad ligament and left ovary into the abdominal wall. At this point a drain entered the ureter, and this ureterostomy apparently had been functioning. On cut section the left kidney showed an acute purulent reaction in the pelvis extending into all the calyces and into the parenchyma. The kidney itself was relatively large and soft. The capsule could be stripped. The demarcation between cortex and medulla was not clearly evident.

The right kidney was likewise greatly enlarged and was surrounded by perirenal fat showing inflammatory changes. This ureter was greatly enlarged and similar to the left ureter. The ureter below the drain was relatively small and contracted, and did not show the inflammatory reaction above noted (Fig. 11A and B). The sigmoid, when opened, showed a tag of necrosing ureter extending into the lumen. Serial sections were made of this juncture because very little could be made out grossly (Fig. 12). The right kidney was slightly larger than the left, weighing 300 grams, and showed all the changes found in the left kidney. In addition there were multiple, small, scattered cortical abscesses.

The parenchyma of the right kidney was distorted by a marked purulent inflammatory reaction extending up from the pelvis. There were many small scattered abscesses throughout the cortex and medulla. A few old scars of an arteriosclerotic nature were present. The acute reaction extended into the capsule. The cortical abscesses and the marked reaction were not seen in the left kidney, but it showed an acute, gangrenous purulent pyelitis with the inflammation extending into the adjacent fat. The right ureter above the ureterostomy showed a zone of acute necrosis superimposed on a chronic inflammatory reaction, and there was an acute peri-ureteritis. This ureter below the ureterostomy was undergoing atrophy and necrosis probably of an infarcted nature. Serial sections were made showing the right ureter entering the sigmoid (Fig. 12). The ureter showed atrophy and necrosis, and, at the point of entrance through the sigmoid, a subacute inflammatory reaction was found extending in all the coats of the bowel with a localized peritonitis. The formation of scar tissue had begun in this region.

SUMMARY AND CONCLUSIONS

The postoperative course of the first patient, and the perfect implantation on one side with normal ureter and kidney (Figs. 9 and 10) found at autopsy 8 months after operation in the second patient, encourage the belief that extraperitoneal drainage by ureterostomy and ureteral catheter, in conjunction with implantation of the ureter into the bowel by the hidden perforating suture

TABLE I.—37 PERSONAL CASES OF URETERO-INTESTINAL IMPLANTATION

	Number of cases	Carcinoma of the bladder		Tuberculous cystitis		Vesicovaginal fistula		Exstrophy of the bladder		Hunner ulcer	Summary	
		16		7		4		9		1	37 cases	
		Dead	Living	Dead	Living	Dead	Living	Dead	Living	Dead	Dead	Living
Modified first method of Coffey	8	1	1		4†				2		1	7
Second technique of Coffey (catheter drainage)	15	4	1			2	1	2 1* 3	3	1	10	5
Third technique of Coffey with extraperitoneal ureterostomy	3	1*	1		1						1	2
Total	26	6	3		5	2	1	3	5	1	12	14
Simple seven suture method	11	1 2* 3	4		1 1† 2		1		1		3	8

*After cystectomy.
†With nephrostomy.

of Coffey, is a method by which bilateral implantation would be safe. The catheter gives free drainage until the suture cuts through. Opposed to this are the imperfect drainage and acute pyonephrosis which was present on the right side in the second patient and the finding at autopsy of the acute ureteritis and the necrosis above the ureterostomy and of anæmic infarction below, in the third patient, a complication which was apparently caused by the retention catheter. This method, therefore, cannot be recommended.

ANTERIOR GASTRO-ENTEROSTOMY BY THE SHORT LOOP METHOD

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PALLIATIVE operations often are indicated in cases of obstructive lesions of the stomach. Posterior gastro-enterostomy has long been the operation of choice in such cases. If its successful completion is not possible, however, some other method must be used. The classical anterior gastro-enterostomy is a poor second choice on account of the frequent complications which follow its use, namely obstruction and ulceration at the site of anastomosis or in the intestine near the anastomosis. Entero-enterostomy in conjunction with anterior gastro-enterostomy lessens the possibility of obstruction but complicates the physiology of the intestinal tract and lengthens the operative procedure in a case which may be already a bad risk. We wish to present a short loop method of anterior gastro-enterostomy which offers the advantages of the posterior gastro-enterostomy and avoids some of the disadvantages of the classical anterior operation.

Recently, in a case of an irremovable obstructive carcinoma involving the mid portion of the lesser curvature of the stomach we found that the lesion had extended into the posterior wall to such an extent that a posterior gastro-enterostomy was not possible. The anterior wall was free from infiltration. It was decided therefore to do an anterior gastro-enterostomy but to make the anastomosis through the mesocolon by a short loop method, and thereby avoid the usual long loop which would have to be drawn anterior to the transverse colon.

The stomach was separated from the transverse colon by ligating the gastric branches of the gastro-epiploic arteries of the stomach in the gastrocolic omentum. This opened the lesser peritoneal sac and freed a major portion of the greater curvature. The transverse colon was then drawn through the abdominal incision and an opening was made in the transverse mesocolon. The anterior wall of the stomach, above the obstruction was drawn through the opening in the transverse mesocolon and one part of a light gastro-enterostomy clamp was applied. The stomach proximal to the clamp was sutured to the margins of the mesocolon, so that after the colon fell to its normal position these sutures rotated the anterior wall of the stomach. In this way, the portion which was superior was brought into a lower plane and the portion to be anastomosed was held in the new position. The jejunum just distal to the ligament of Treitz was located and placed in the other portion of the gastro-enterostomy forceps. Then the antiperistaltic anastomosis was completed. The upper portion of the jejunum midway between the mesenteric and the antimesenteric border rather than the antimesenteric segment was the portion placed in the clamp. This avoided a rotation of the intestine in the axis of its lumen following anastomosis. Finally, the anterior wall of the stomach above the anastomosis was sutured to the transverse colon along the line of the gastrocolic omentum from which the greater curvature of the stomach had been separated.

The patient made an uneventful recovery. He had no nausea or vomiting and to date, 8 months after operation, has had no untoward symptoms. Deep X-ray therapy was instituted 3 weeks after the operation was performed.

The technique of the operation by steps is as follows:

1. An upper abdominal incision following the method advocated by Sloan is used.

2. The greater curvature of the stomach is freed from the transverse colon by ligating the gastric branches of the gastro-epiploic arteries directly below the chosen site of anastomosis, thus giving entrance to the lesser peritoneal cavity. (Fig. 1).

3. The colon is drawn out of the incision, tensing the mesocolon which is opened between the colic vessels sufficiently to allow the anastomosis to be made.

4. The anterior wall of the stomach is sutured to the upper margin of the opening in the transverse mesocolon with interrupted chromic catgut sutures placed about three-quarters of an inch apart (Fig. 2).

5. The anterior wall of the stomach is drawn through the opening of the mesocolon and held by a light gastro-enterostomy clamp. Then the remainder of the opening in the transverse colon is closed by suturing it to the stomach proximal to the clamp.

6. The jejunum is located just distal to the ligament of Treitz and a short loop is placed in the anastomosis clamp in position for antiperistaltic anastomosis (Fig. 3A).

7. The anastomosis is closed by three continuous sutures of chromic catgut, the first layer

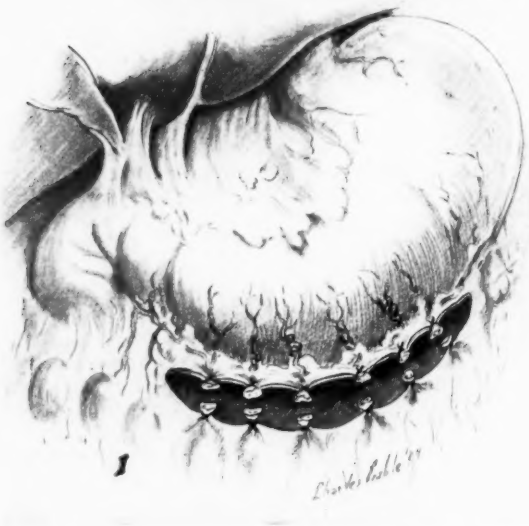

Fig. 1. Short loop anterior gastro-enterostomy. Opening into lesser peritoneal cavity through gastrocolic omentum by ligation and section of gastric branches of gastro-epiploic arteries.

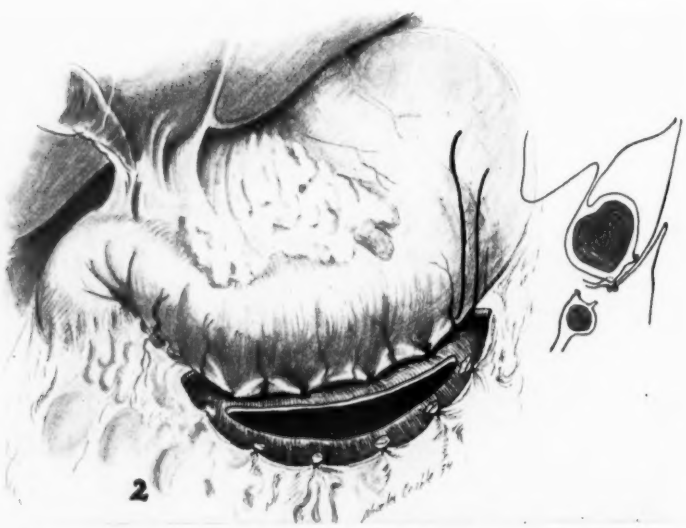

Fig. 2. Short loop anterior gastro-enterostomy. Suturing of stomach to upper margins of opening in transverse mesocolon.

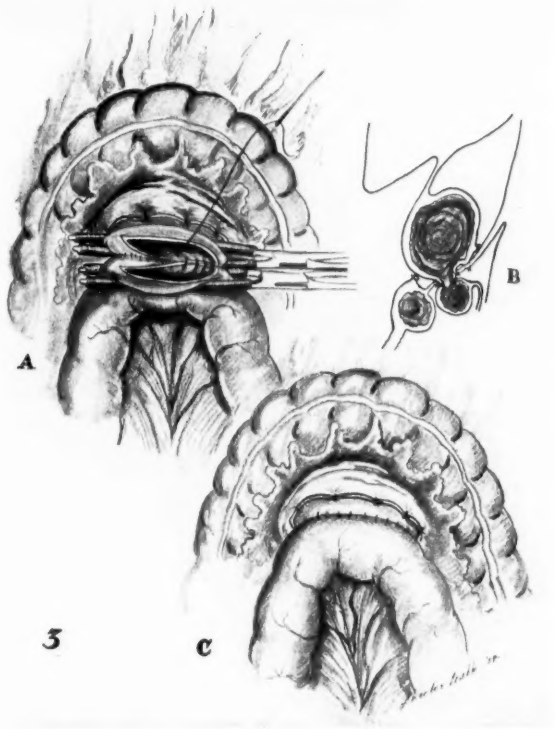

Fig. 3. Short loop anterior gastro-enterostomy. A, Short loop of jejunum placed in anastomosis clamp. B, Relative position of jejunum to stomach after completion of operation. C, Appearance of completed operation from below—similar to posterior gastro-enterostomy except that the anterior instead of the posterior wall of the stomach is anastomosed to the jejunum.

including the serosa and the muscularis, the second layer including the serosa, muscularis, and submucosa, and the third layer including all three layers as an over-and-over hæmostatic stitch (Fig. 3 B and C).

8. The area on the colic side of the gastrocolic omentum first detached from the stomach is then sutured to the anterior wall of the stomach.

9. The abdomen is closed without drainage.

CONCLUSION

A technique for a short loop method of gastro-enterostomy is presented as a substitute for the classical anterior gastro-enterostomy in those cases in which a posterior gastro-enterostomy is not technically possible.

We believe that the method which is here presented offers the advantages of a short loop posterior operation and that it avoids the disadvantage of the possible complications which usually accompany the long loop anterior gastro-enterostomy.

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SLOAN, G. A. A new upper abdominal incision. Surg., Gynec. & Obst., 1927, 45: 678-687.

MESENTERIC CYSTS[1]

A Brief Discussion and Report of Three Cases

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PROBABLY the first cyst of the mesentery was described by Benivieni, a Florentine anatomist of the sixteenth century. He discovered the tumor at autopsy and recorded it as an anatomical curiosity. The lesion is sufficiently uncommon to arouse considerable interest whenever encountered. Possibly about 500 cases are recorded in the literature. In 1932, in a very fine review, Warfield found a total of 129 reported cases since 1920. Carter, in 1921, estimated that between 200 and 300 cases were recorded, while Flynn in 1930 thought the literature contained no more than 300 cases at the most. Judd states that in 820,000 admissions to the Mayo clinic hospitals, only 25 cases of mesenteric tumor were found, and only 8 of these were cysts. Therefore, in this clinic, the incidence of mesenteric cyst was less than 1 per 100,000 admissions, while a tumor of some kind was found in the mesentery in 1 of some 35,000 cases. The Massachusetts General Hospital has a record of only 6 cases from 1900 to 1926. The University of California Hospital has a record of 1 cyst and 1 tumor of the mesentery, in 93,511 admissions, since 1906. The Children's Hospital, Los Angeles, has 3 cases recorded, all in females, and all occurring in the last 3½ years, in a total of 12,425 admissions during the same period. Los Angeles General Hospital has a record of 1 case in 188,921 admissions in the last 5 years. Lane Hospital reports 3 cases from 1911 to 1933 in approximately 77,000 admissions. The New Harborview Hospital, Seattle, has no record of a mesenteric cyst in 27,000 admissions, and approximately 12 surgeons and pathologists in Seattle have no record of a case.

Warfield found in 129 cases the sex distribution as follows:

	Cases
Female	71
Male	37
Unknown	21
	129

Nine of the cases were in infants under 1 year of age, and 21 were in children from 1 to 10 years of age. The age group from 30 to 40 showed the greatest incidence, 24 cases. I have encountered 3 cysts of the mesentery on my own service of about 900 surgical admissions, during the last 2½ years, or 1 in every 300 cases. Two were cases of intestinal obstruction, with the cysts as the etiological cause, and the other was a small cyst, possibly symptomless, encountered during an operation for recurrent appendicitis. All occurred in males, all were undiagnosed before operation, and all were found in the mesentery of the ileum less than a meter from the ileocæcal junction. In the literature, women seem to be affected about twice as often as men. Statements appear that the diagnosis is never made before operation. However, correct pre-operative diagnoses were made by Haworth, 1920; Bertolini, 1921; and later by Aloi, in 1927; and Finucci 1930; and others.

The history of these cysts has been well divided into four periods: (1) 1707 to 1850, when the lesion was only discovered at autopsy; (2) from 1850 to 1880 when an occasional cyst was operated upon, but there were no recoveries; (3) from 1880 to 1900 when operation was beginning to be followed by a few recoveries; and (4) from 1900 on, when the condition was becoming definitely known, was more successfully treated, and occasionally diagnosed before operation. Possibly it is the rarest tumor of the abdomen. It may occur at all periods of life from infancy to extreme old age. Textbooks refer very briefly or not at all to the condition, and many surgeons of wide experience have never encountered a case.

The cysts may vary in size from less than a centimeter in diameter to truly huge tumors that fill the entire abdomen; thus they may be too large or too small to diagnose easily. About 70 per cent appear in the mesentery of the small bowel and the remaining 30 per cent are found in the mesentery of the large bowel or other mesenteries. Warfield's 129 cases were located as follows:

	Cases
Small bowel mesentery	61
Large bowel mesentery	50
Gastrohepatic mesentery	1
Appendix mesentery	2
Unknown or unstated	15
	129

They are usually oval, round, or bosselated, may be unilocular, bilocular, or multilocular, and

[1] Read before the General Surgery Section of the California Medical Association, Riverside, California, April 30–May 3, 1934.

may be filled with chyle, clear serous fluid, hæmorrhagic fluid, clotted organized blood, or other material. Some contain cholesterin crystals. Many times the walls are greatly thinned and any lining membrane the cyst may have possessed earlier has been destroyed through pressure atrophy. Likewise the entire wall may give no definite clue as to the origin of the lesion. Oftentimes the contents are no more valuable as far as etiology is concerned.

DEFINITION

A true mesenteric cyst must occur between the leaves of the mesentery or under the serosa of the gut, and must not be a retroperitoneal cyst, although it may have originated from sequestered material which has migrated from retroperitoneal embryological organs to a position between the leaves of the mesentery. Cysts occurring retroperitoneally or under the peritoneum of the abdominal wall may have a similar origin but are not mesenteric cysts by location. Cystic malignant disease of the mesentery should be classified under malignant tumors, as very rarely is there a record of a mesenteric cyst becoming malignant; yet, Deaver suggests that such is a possibility, as branchial cysts sometimes become malignant.

ETIOLOGY AND CLASSIFICATION

It seems impossible to determine accurately the etiology of many of these cysts, and so many classifications have been offered that the affair is confusing. Since quite a number occur in young children, it is fair to assume that many of them are embryological in origin. A classification follows:

1. *Embryocystomata or embryonic inclusion tumors.* (a) Those derived from sequestered remnants of the wolffian body or its duct, or the muellerian duct. In other words it seems possible that portions or remnants of the urinary or genital embryological organs could become detached, migrate to a position between the leaves of the mesentery, retain or develop a blood supply, and develop into cysts. Pararenal cysts in all probability have origin from the wolffian body but nevertheless they retain their position near the kidney.

b. Cystic dermoids and teratomata. The genital glands in both sexes develop behind the peritoneum as do other of the abdominal organs, and the same theory holds true.

c. Dermal inclusions. This does not seem to account for many cysts although a dermal sebaceous cyst has been reported in the mesentery, by Swartley in 1927. Bartlett, in 1923, suggested this as an origin.

d. Enterocystomata, or cysts developing from misplaced intestinal tissue. Here sequestrations from the intestine, fetal intestinal diverticula, or from the omphalomesenteric duct (vitelline) become displaced to positions between the leaves of the mesentery. In this manner cysts from intestinal remnants may occur anywhere along the digestive tract, while those from the vitelline duct occur principally in the mesentery of the ileum near its termination. Meckel's diverticulum represents the failure of the intestinal end of this same duct to become completely obliterated. Umbilical cysts may represent the failure to close of the other end of this duct. Miller reports a case of a 4 day old baby in which the wall of the cyst was, in one portion, directly continuous with the wall of the jejunum and the histological structure of the cyst wall resembled rather accurately the wall of the bowel. Here seems proof of a mesenteric cyst that owed its origin to sequestration or diverticulation of embryonic intestine. Minute intestinal diverticula and cysts are not infrequently found in rabbits.

2. *Chylous cysts.* Ewing suggested that these cysts were chyle angiomata, which arose either from congenital or acquired obstruction to the mesenteric lymphatics and lacteals, carrying the chyle from the intestines to the receptaculum chyli. However, Dowd thought that the chylous nature of some of these cysts was due to an effusion of chyle into a preformed cyst, especially since the very rich anastomosis of the mesenteric lymphatics seems to preclude that obstruction to even many channels would form a cyst.

3. *Bacterial of parasitic cysts.* It seems possible that the necrosis and liquefaction in the enlarged mesenteric lymph glands of tuberculosis, typhoid fever, or bacterial infection of other nature might result in cyst formation. There is some evidence to support this. Parasitic, hydatid, or echinococcus disease has been responsible for cystic change in the mesentery.

4. *Traumatic cysts.* These may be filled only with blood and result from operative or other trauma to the mesentery, resulting in hæmorrhage between its layers, with or without subsequent serous change. Genkin in 1928 reported a chylous cyst in the mesentery formed around a gauze sponge left in the abdomen during a previous operation.

5. *Angiomata of blood or lymph vessels.* Very few cysts of the mesentery seem to be true lymph or hæmangiomata, and probably only a few cysts have this origin. However, in the past, competent

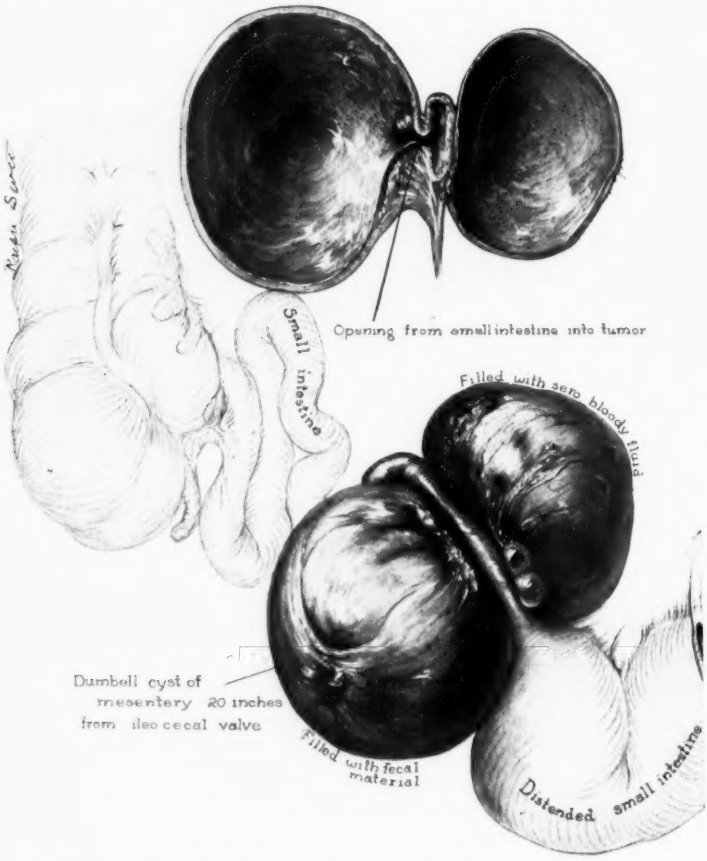
Opening from small intestine into tumor
Small intestine
Filled with sero bloody fluid
Dumbell cyst of mesentery 20 inches from ileo cecal valve
Filled with fecal material
Distended small intestine

Fig. 1. Case 1. Drawing of cysts as they appeared in the abdomen and also on cross section, showing the mechanism of the obstruction and the recent perforation of the ileum into one locule, converting it into an abscess.

authorities have thought many of these cysts were a form of lymphangioma.

6. *Gas cysts.* Gas cysts are rare, usually symptomless, and nothing definite is known as to etiology. They may be multiple. Possibly they do not belong in a classification of this sort, as they seem fundamentally different.

SYMPTOMS

There are no definite pathognomonic findings applicable to the general run of cases. Most of them must be symptomless early in their development, and even until they are quite large, when the patient may note enlargement or a growth in the abdomen only. Usually the provisional diagnosis before operation is not correct. However, a rounded, freely movable, cystic, nontender abdominal mass, should suggest the possibility of a mesenteric cyst. According to several writers, the mobility is most striking in the transverse direction. Sometimes a belt of resonance can be percussed over the tumor where the intestine passes over it. Ruling out connections with the lumen of the bowel, and other findings made with the barium meal or enema, and fluoroscopy and films, should be valuable. Pressure from a cyst may give symptoms, depending upon which intra-abdominal organ it is encroaching upon. In one of my cases initial urinary symptoms were possibly caused by pressure of the cyst upon the urinary bladder. When complications arise the symptoms may be almost anything,

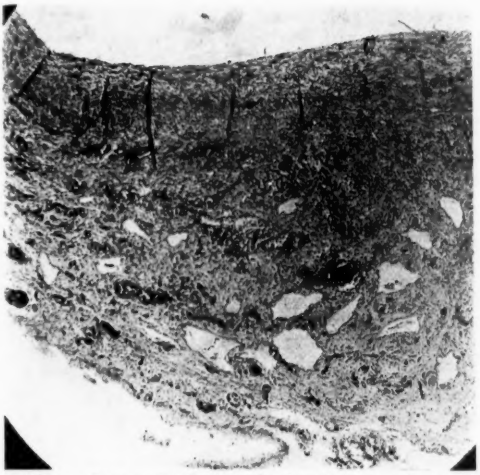

Fig. 2. Case 1. Wall of smaller locule (contents sterile) showing an organized granulation tissue and some large endothelial lined spaces.

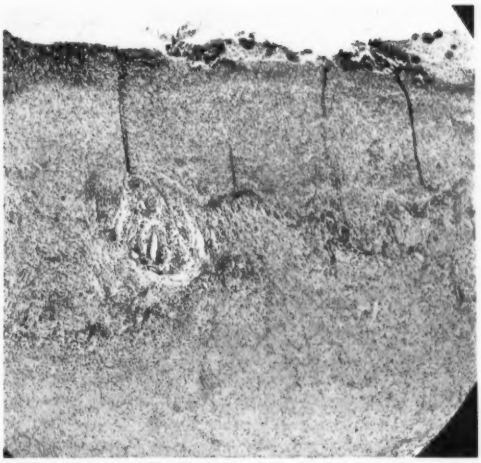

Fig. 3. Case 1. Wall of larger locule (abscess due to intestinal perforation) showing inner surface which has a fibropurulent exudate.

are usually very pronounced, and about 40 per cent of all complications are intestinal obstruction, either acute, chronic, or intermittent in type. When acute complications ensue, the differential diagnosis would have to include almost every cause for the acute abdomen, so varied may be the manifestations. It is well to bear in mind that an etiologically obscure but definitely acute abdomen, should be explored without much wasted time. The main point, so far as the preoperative diagnosis is concerned is to bear in mind the possibility of a mesenteric cyst.

PROGNOSIS

Atchley states the mortality to be 35 per cent for the cases as a whole. As previously mentioned, intestinal obstruction brings about 40 per cent of the cases to medical attention and in this group the mortality is variously stated to be from 30 to 50 per cent. Warfield, in his 129 reported cases gives a known, more or less immediate mortality of 20 per cent. In 23 of this series of cases the patient was able to leave the hospital but no follow up record was available. Of course the prognosis depends largely upon the time of diagnosis and operation, type of operation found necessary, size, location, attachments and complications present and the age and general condition of the patient.

COMPLICATIONS

These in the main are intestinal obstruction, with or without gangrene of the bowel wall, volvulus (as present in one of my cases), intussusception, adhesions, or attachments to organs, hæmorrhage into a cyst, rupture of a cyst into the abdominal cavity, rupture of the cyst into the bowel or vice versa, causing acute inflammation and abscess formation in the cyst (as in another of my cases), perforation of an infected cyst with

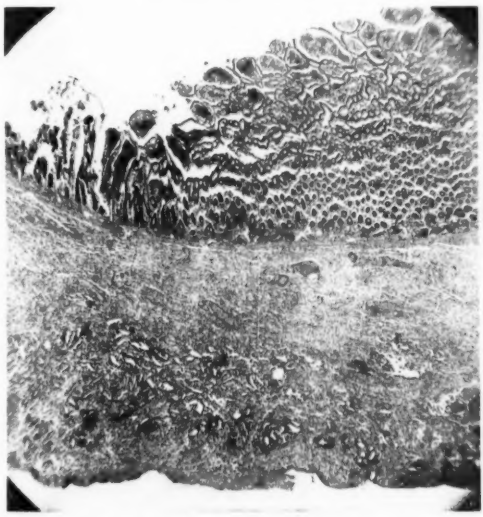

Fig. 4. Case 1. Shows the granulomatous reaction in the wall of the small intestine near the perforation. The crystals represent cholesterol, probably due to inspissation or fat necrosis.

Fig. 5. Case 2. Showing tumor and volvulus and mechanism of the intestinal obstruction; also removed specimen.

peritonitis, ileus, and gastric dilatation, and the complications that may ensue after abdominal surgery.

TREATMENT

This part of the subject seems to be rather definitely settled. The treatment is purely surgical, and in order of desirability is as follows:

1. Enucleation, if possible, without jeopardizing the viability of the gut by too much damage to its blood supply. This procedure is accompanied by about 9 per cent mortality.

2. Enucleation or resection accompanied by removal of a portion of bowel and anastomosis. Mortality is 25 to 30 per cent.

3. Marsupialization or incision and drainage. This method should be used only when it is out of the question to do otherwise, due to size, location, adhesions, or poor condition of the patient. The mortality is about 16 per cent. Cure is not always accomplished by this method and drainage persists for some months as a rule.

4. Aspiration seems to me to be a very unsatisfactory method of treatment. It has its place only as a means of diagnosis (with abdomen open) or to reduce the size of cyst preparatory to removal.

CASE REPORTS

CASE 1. Master, A. P., white American boy, aged 6 years, was referred to me on February 16, 1933, with the following history. He had pain in the right side of abdomen, nausea, vomiting and obstipation. The child had gone to school the previous day and returned home in the afternoon complaining of not feeling well all day. He complained of sharp pains all over abdomen, mostly in the right side. Nausea soon developed accompanied by vomiting, which continued throughout the night. A physician was consulted the next morning who diagnosed acute appendicitis and advised taking the child to the hospital at once. This was not done until 6:30 the same evening, after the child had been given castor oil and enemata, with no results other than to increase the pain and vomiting.

The past history was uneventful except for measles and for the fact that during the last 6 months at intervals of 3 or 4 weeks, the child had had sudden attacks of abdominal pain, with vomiting, lasting only a few hours, after which the child again felt perfectly well. The mother says the child was constipated before these attacks and that laxatives and enemata gave relief. She thinks he had no elevation of temperature.

Physical examination revealed a child acutely ill. He lay in bed with thighs flexed, and cried with pain if moved. The abdomen was moderately distended and to inspection it seemed to be more prominent in the right lower portion than elsewhere. The abdomen moved very slightly with respiration in the upper portion, and palpation determined the presence of a round, exquisitely tender, non-movable mass as large as a baby's head in the right lower quadrant. The right side of the abdomen was rigid, to such a degree that it was impossible to outline the mass accurately. It did not involve the kidney region. It could be felt by rectal examination. Except for evidences of dehydration, the rest of the physical examination elicited normal findings.

Laboratory examination of voided urine showed color amber, clear; reaction, acid; specific gravity, 1024; albumin and sugar, negative; 1 plus acetone; microscopic examination normal.

Blood examination showed hæmoglobin 80 per cent, Salhi, red blood cells, 4,700,000; white blood cells, 20,000; polymorphonuclears, 84 per cent.

Diagnosis: (1) Abdominal tumor with intestinal obstruction and peritonitis; (2) appendiceal abscess with obstruction and peritonitis.

Operation. After adequate preparation the abdomen was opened, under nitrous oxide-oxygen-ether anæsthesia. Dilated coils of intestine, a bloody serous exudate, which was cultured, and a bilocular cystic, bluish tumor arising from the mesentery of the ileum about 20 inches from the ileocæcal valve, and freely movable, were found. The tumor was easily delivered into the wound. Each locule was the size of an orange, about 9 centimeters in diameter, and one was covered with a glistening peritoneum and the other with a red inflamed peritoneum. The neck was broad and connected the two locules just under the mesenteric border of the gut in such a manner that the tumors so compressed the lumen of the ileum that almost complete obstruction existed. The appendix was normal.

An attempt was made to enucleate the tumors, but in so doing the inflamed locule was punctured in an area adherent to the intestine, through which the ileum had perforated into the cyst, forming an abscess within the cyst, and a few drops of foul smelling fæcal material escaped. The rent was at once closed by suture and a resection of 3 inches of ileum, cysts, and a V of mesentery done with end-to-end anastomosis following. Because of rupture during operation a small Penrose drain was inserted down to the anastomosis and the abdomen closed in layers. The patient made an excellent recovery. There was no vomiting, beyond the first 12 hours after surgery. Fluids were maintained by hypodermoclysis of normal saline and 5 per cent glucose retention enemata for the first 48 hours. The drainage from the abdomen was only moderate, the drain was removed on the third day after operation. The bowels were controlled easily with mineral oil. A temperature rise occurred on the sixth day after operation from a small abdominal wall abscess and on the twenty-second day from an upper respiratory infection. Both were easily controlled. The patient left the hospital on the thirty-first day after operation, with the wound completely healed and the bowels regular without laxatives. He has been followed at intervals for over a year, is growing rapidly, doing well in school, and has no complaints of any kind. No incisional hernia developed.

Pathological diagnosis by Dr. Edwin I. Bartlett: Undifferentiated cysts of the mesentery.

This tumor should have been easily movable, but the acute abdomen and obstruction rendered the abdomen so spastic that this point could not be made out before operation. Had not the perforation into one cyst locule been present we probably could have performed the less dangerous operation of enucleation instead of resection with anastomosis of bowel. Probably the perforation occurred the day or night before operation, producing the fever, leucocytosis and swelling in the cyst locule, and much more complete intestinal obstruction than the child had had with the previous attacks.

The contents of one locule were distinctly serous. The contents of the locule into which the gut had perforated were cloudy, purulent, and fæcal smelling. Cultures of this fluid gave abundant growth of intestinal organisms, while cultures of the other locule were negative. The contents were not chyle like. Because of the location this could have been a cyst originating from either an intestinal diverticula or from the omphalomesenteric duct or from lymphatic ducts or lymphangioma.

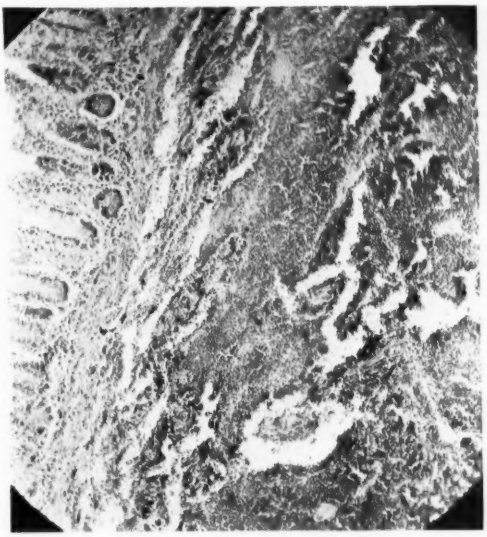

Fig. 6. Case 2. Representative section of the cysts and also of the adjacent bowel, showing extravasated blood in the mesentery, and extending to muscularis and muscularis mucosæ of the damaged loop of bowel.

CASE 2. Mr. B. C. M., adult white male, single, aged 53 years, was admitted to the hospital at 2:15 p.m., on August 19, 1933. He had generalized intermittent abdominal pain, vomiting, obstipation, urinary difficulty, and persistently distended abdomen for the past 48 hours. He began feeling ill 3 days ago and could not pass urine. He went to a doctor who catheterized him easily and obtained only a few ounces, after which he had been able to void small amounts. Since then he had not been seen by a physician until just before admission to the hospital. Two days ago the pain became worse and patient started to vomit. Several enemata and a laxative failed to move the bowels. He has had considerable abdominal pain of an intermittent character, which he says extends over the whole abdomen and he thinks it is worse about the umbilicus and over the bladder region. He feels gas moving about in the abdomen and at these times the pain is most severe. He has vomited persistently for 48 hours and the bowels have not moved. He did not vomit any fæcal smelling material.

Patient has always been well and strong except for a right inguinal hernia repaired 15 years ago and an appendectomy performed 12 years ago. The hernia has not recurred since and he left the hospital in 12 days after his appendectomy with the wound fully healed. He has noted a gradual increasing constipation for the last few years, but thought little of it. He has had no urinary difficulty whatsoever until the present illness.

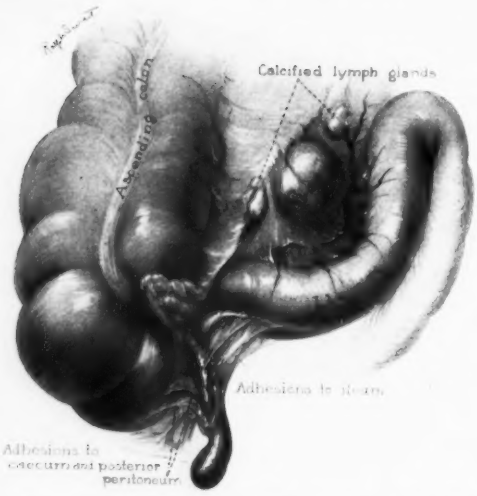

Fig. 7. Case 3. Drawing showing the size and location of the cyst, the two calcified mesenteric glands, and the appendix.

The family is negative as far as the present illness is concerned.

Physical examination: Patient is obviously very ill. The skin, tongue, and mucous membranes are dry and dehydrated; pulse 108 per minute; temperature 99.8 degrees F.; and respirations 22 per minute. The heart seems slightly large to percussion and a non-transmitted murmur is heard at the apex. Tonal quality is good and there is no evidence of decompensation. Blood pressure is 124-72. Patient vomited about a pint of bile stained foul stomach contents during the examination. The abdomen is greatly distended and tense, the percussion note is tympanitic throughout. There is moderate tenderness and muscle spasm over the lower abdomen, and no masses or organs are palpable, owing to the distention. With the stethoscope an occasional peristaltic movement can be plainly heard, corresponding to the patient's spasms of pain. The bladder is not distended. Catheterization is easily done and yields about 5 ounces of clear dark urine. The rectal examination reveals what appears to be distended coils of gut deep in the pelvis or a soft, only slightly tender mass. The prostate is small and sphincter tone good. The rest of the examination revealed nothing of note.

Laboratory examination showed the urine dark, clear in color; specific gravity, 1028; reaction, neutral; trace of albumin, no sugar, 1 plus acetone. Microscopic examination showed an occasional hyaline cast, occasional pus and epithelial cell. The specimen was centrifuged.

Blood examination showed white blood cells, 8,000; red blood cells, 4,600,000.

Diagnosis: Acute intestinal obstruction, probably from adhesive bands about terminal ileum from previous laparotomy.

Procedure: Patient was put to bed, and several hours were spent preparing him for operation. He received 2500 cubic centimeters of salt solution subcutaneously; enemata, without results; stimulants and gastric lavage. Under low spinal anæsthesia laparotomy was performed. The entire abdomen was filled with tense, distended coils of ileum, quite reddened in appearance, so we determined that the obstruction was quite low down. Inserting the hand into the pelvis a tumor mass was found deep down against the bladder, and feeling cystic and lobulated. It was easily delivered into the wound. The tumor was seen to be a multilocular cyst of the mesentery of the ileum, about 24 inches from the ileocæcal junction, 4½ times 2½ inches in diameter, and with the cyst locules very closely encroaching upon the gut and one or two bulging past the antimesenteric border. There was a complete volvulus or torsion of the cyst and involved intestine, i.e. one twist of the mesentery, causing a complete intestinal obstruction and a pregangrenous condition of the involved gut. Fortunately the volvulus was not tight and very little of the mesentery was hæmorrhagic and thrombosed. The distended coils of gut were so in the way that an ileostomy was done 8 inches above the obstruction, using a No. 16 catheter and by very gentle suction a great deal of liquid material and gas was removed. Resection of the cysts, about 5 inches of bowel and the injured mesentery was done followed by an end-to-end anastomosis. The abdomen was closed in layers about a small drain and the ileostomy tube.

The patient was returned to the ward in fair condition, blood pressure 100 systolic and fully conscious and rational. The ileostomy tube was allowed to drain every alternate hour, the patient was digitalized, 3000 cubic centimeters of normal saline was given subcutaneously per 24 hours and 200 cubic centimeters of 3 per cent of saline given intravenously the first 3 or 4 days. Retention enemata of 6 ounces of 5 per cent glucose were given every 4 hours. (Not only does this last supply fluid, but the irritation it causes in the lower bowel stimulates peristalsis higher up, and is of material aid in the after-care of a case of this kind.) There was considerable purulent drainage from the abdomen, and the drain was shortened daily and removed completely the fifth day after operation. The bowels were moved daily with a small enema. The first bowel movement unaided by an enema occurred on the sixteenth day after operation. The ileostomy tube was clamped off permanently on this day and was removed on the nineteenth day after operation. The fistulæ closed rapidly and the patient left the hospital on the twenty-eighth day after operation with his wound completely healed except for a non-draining granulating area at the site of emergence of the catheter. His general condition was good and his bowels were moving well on ½ ounce mineral oil twice a day. He was seen 1 week later feeling fine, wound completely healed, appetite and bowels were normal. This patient was last seen on December 20, 1933, about 3 months after operation when he had gained about 20 pounds in weight, and his bowels were regular without laxatives or mineral oil. He had no complaints.

Probably this cyst resulted from trauma to the mesentery possibly during the appendectomy 12 years previously. There is no history of other abdominal trauma. There is no proof that it is a hæmangioma.

Diagnosis by Dr. Edwin I. Bartlett: blood cysts of the mesentery.

CASE 3. Mr. L. H. M., white American male, aged 46 years, was admitted to the hospital March 28, 1933. Patient complained of pain and discomfort in the right lower abdomen, at intervals for the past 2 years. About 2 years ago the patient began to be conscious days at a time of pain of an aching character in the right lower quadrant, aggravated by eating and working. He sometimes felt nauseated, but never vomited. After a few days the pain would leave and not occur for 2 or 3 weeks. The last few months the condition has been worse and he comes to the hospital for relief. There is no gall bladder pain, no ulcer

history, no urinary symptoms, and the pain does not radiate. Patient admits having syphilis in 1906. He was treated at intervals until 1919 and since then the Wassermann reaction every year has been negative. No gonococci were noted. He has never been ill otherwise. He has always been a native of California.

The family history is negative for tuberculosis or chronic disease.

Physical examination: temperature, pulse, and respiration were normal. Head and neck were negative, heart and lungs normal. The blood pressure was 130–84. The abdomen was flat, moved well with respiration, there were no organs or tumors palpable, and no rigidity, but very definite point tenderness to deep palpation over the appendiceal area. Rectal examination revealed only a few small hæmorrhoids, a slightly enlarged prostate, and good sphincter tone.

A plain film of the abdomen revealed normal kidney shadows, and in the appendiceal area and above the brim of the pelvis were two shadows, like stones, 2 by 1 and 1 centimeter in diameter, respectively. A barium enema revealed a normally filling and appearing large bowel, a competent ileocæcal valve, and a poorly filling appendix which was not very movable and was very tender to deep palpation. The two shadows observed in the plain film, were seen about 3 inches above and a little medial to the appendix, and were freely movable, and not tender. We thought they were calcified mesenteric glands.

Spinal fluid and blood Wassermann reactions were negative. The urine was negative, the hæmoglobin 82 per cent Salhi, red blood cells, 4,600,000; white blood cells, 6,900.

Diagnosis: recurrent appendicitis, calcified mesenteric lymph glands.

Operation revealed a short, bulbous, chronically inflamed appendix, adherent to the cæcum and ileum. The two shadows were found to be calcified lymph glands in the mesentery of the ileum about 3 or 4 inches from the ileocæcal junction, one near the root of the mesentery the other nearer the gut. Between the two, was a thin walled cyst, of the mesentery, about 3 centimeters in diameter. The glands and appendix were removed. The cyst was shelled out, but in so doing the very thin wall ruptured, a colorless thin fluid escaped, and the wall was subsequently lost, so that we were unable to study it. Convalescence was uneventful. The patient was discharged on the eleventh day after operation. He was last seen March 6, 1934, almost a year after operation. He has had a complete relief of symptoms. Crushed sections of the nodes revealed nothing but calcium. Pathological diagnosis of the appendix was normal. Only one cross section was examined, and the organ was not normal grossly. This cyst might have resulted from liquefaction of an infected lymph node, especially since on either side was a calcified node. Could the appendix have caused the lymphadenopathy? No other cause was found.

SUMMARY AND CONCLUSIONS

The first cyst of the mesentery was found at autopsy in the sixteenth century. The history of the tumors is divided into four periods. Possibly four or five hundred cases have been reported in the literature. It is probably one of the rarest of abdominal tumors. The etiology, correct classification, and pathology are obscure. The mortality rate is higher than it should be, and the diagnosis is uncertain before operation; both should be improved. Possibly if we would bear in mind the possibility of mesenteric cysts in obscure abdominal lesions we would diagnose more cases before operation. Complications are disastrous, as a rule, often give the first symptom and thus obscure and cloud the diagnosis. Uncomplicated cases, and most cases taken early should have a relatively low mortality. Three personal cases are reported fully. All recovered. Two are known to be alive and perfectly well more than a year after operation, and the third was doing very well when last seen 3 months after surgery. Two of the cases were obscured by grave and serious complications, and the third was a small cyst, possibly symptomless. The etiology was not determined exactly in any case. A possible classification of these lesions is presented. The subject is extremely interesting, from the standpoint of etiology, symptomology, and diagnosis, and it should be emphasized that it be considered more often in the differential diagnosis of intra-abdominal disease.

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TERATOMA OF THE SPINAL CORD[1]

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TERATOMATA of the spinal cord are among the rarest of tumors. The following case, in which such a tumor was found, is of additional interest because of the difficulty in differential diagnosis which it presented, and because it so clearly illustrates the amazing ability of the spinal cord of a child to recover from severe compression.

CASE REPORT

R. B., onset with irritability and anorexia at 16 months of age; slowly developing paraplegia with pain in left ankle; examination revealed a paraplegia without sensory change; lumbar puncture demonstrated a complete subarachnoidal block with Froin's syndrome; lipiodol injected; roentgenographic changes in lower dorsal and lumbar spine; operation at 2¾ years of age, removal of teratoma of spinal cord; recovery.

R. B., unit No. 83815, male, was born on June 20, 1931. He was referred by Dr. R. W. Carpenter of Geneva, Illinois, and first admitted on the pediatric service of the University of Chicago Clinics on June 8, 1933, with complaints of "failure to gain weight and weakness of the left leg."

This child was the last of four pregnancies. The first pregnancy terminated in a premature birth, the infant died shortly thereafter. The second child was a "blue baby" and is at present alive and apparently well (although not examined) at 6 years of age. The third pregnancy terminated in an abortion. The remainder of the family history has no obvious relation to the condition of this child. R. B. was normal at birth and weighed 7 pounds, 4 ounces. Except for an attack of impetigo shortly after birth his infancy was uneventful. He walked at 1 year of age and weighed 21 pounds. There were no other illnesses.

In the latter part of October, 1932 (age 16 months), he began to cry more frequently, became very irritable, and it was difficult to get him to eat. At about the same time it was noted that the neck was becoming stiff. This persisted for about 6 weeks. He had no fever, there was no vomiting or diarrhœa. This illness continued unchanged and in November he was confined to bed where he remained until March, 1933. During this period he was unable to raise his legs, but in March he began to stand and walk a little with aid. He complained that he did not want to ride in an automobile because "something hurts me," and he complained of pain in the back on being moved. He was said to have continued to improve but when first seen in June, 1933, he was unable to stand or walk alone. He no longer cried and seemed much less irritable. In a note Dr. Carpenter stated that he had noted a weakness of the left leg which had first appeared a short time after the onset of the illness in October.

Examination on June 8, 1933 revealed a very thin, pale child. He seemed quieter than a normal child of his age. Except for the malnutrition the chest and abdomen seemed entirely normal. Neurological examination revealed no abnormalities of the cranial nerves, the upper extremities seemed quite normal but the left leg showed slight generalized wasting, particularly in the gastrocnemius and gluteal muscles. The left foot was everted. There was also definite weakness of the left lower extremity. The tendon reflexes of the upper extremities were diminished but equal. In the lower extremities the knee jerk was less active on the left than the right and the left Achilles tendon reflex was absent; whereas it was present on the right. The abdominal reflexes were present and equal on the two sides. Babinski's sign was not present. No sensory disturbance could be found.

At that time the child weighed 9 kilograms (19.8 pounds). Laboratory examination revealed a normal urine, a blood count of 3,900,000 erythrocytes, 8,000 leucocytes, and 78 per cent hæmoglobin. The Wassermann and Kahn tests on the blood were negative, as was the test with tuberculin of 1:1000 dilution. Roentgenological examination was made of both lower extremities, the pelvis, the lower lumbar spine and sacrum, and the stomach. All were reported as normal.

It was felt that his condition was the result of an acute anterior poliomyelitis. The parents were given instructions as to diet and physiotherapy and the patient was discharged.

On January 15, 1934, the mother wrote that the child had improved slightly, had a better appetite, and had gained a few pounds. However, in December, 1933, he had

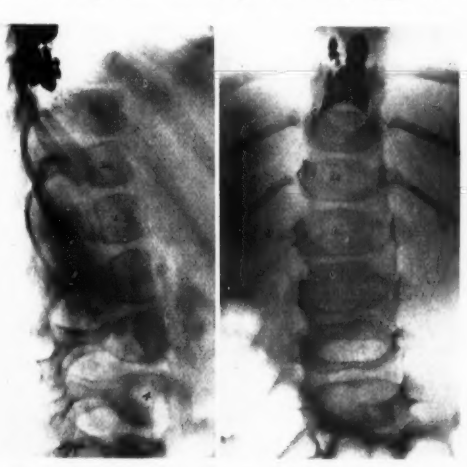

Fig. 1. Lateral roentgenogram of the lower thoracic and lumbar spine. The lipiodol arrested at the upper border of the eleventh thoracic vertebra is seen above. The anteroposterior diameter of the spinal canal is greatly increased at the level of the first, second and third lumbar vertebræ as compared with the normal canal at the fourth, and fifth lumbar vertebræ. The pedicles of the first lumbar vertebra are decalcified.

Fig. 2. Anteroposterior view of the spine. The lipiodol resting upon the superior curved surface of the tumor is present above. The canal from the twelfth thoracic to the third lumbar inclusive is grossly widened, in fact it is wider than the bodies of the vertebræ at this level. The pedicles of the twelfth thoracic, first, second, and third lumbar vertebræ particularly the middle two, are markedly flattened as compared with the normal pedicles of the fourth and fifth lumbar vertebræ.

[1]From the Division of Neurology and Neurosurgery of the University of Chicago.

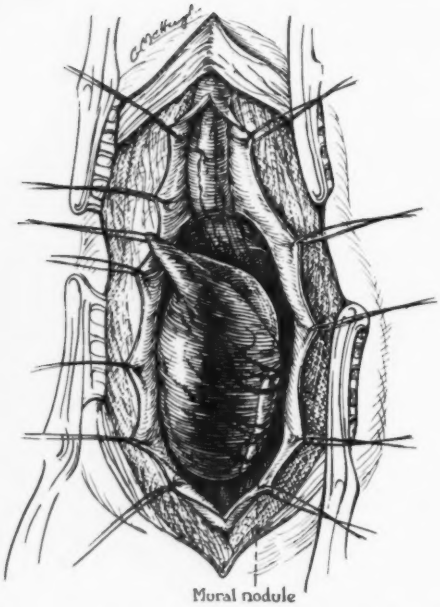

Fig. 3. Drawing of tumor made at the operation. The spinal canal is grossly dilated. The cyst has been drawn to one side and rotated so as to reveal the flattened spinal cord beneath it and the mural nodule in its lower part which was attached to the spinal cord.

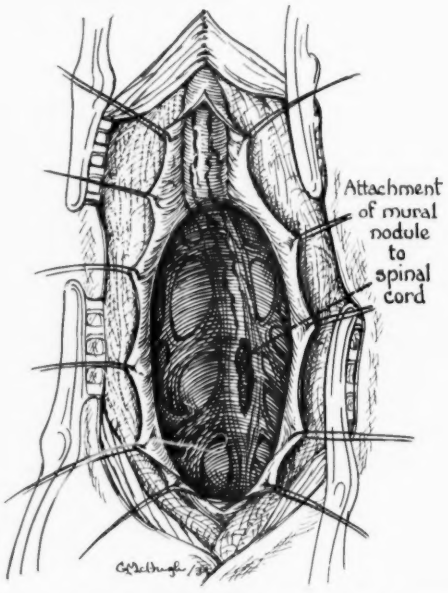

Fig. 4. Drawing of spinal canal after removal of tumor showing the dilatation of the canal and the severe compression of the spinal cord and its roots. The defect in the posterior columns from which the mural nodule was removed is shown.

begun to complain of "severe pains in the left leg, particularly in the region of the ankle." These pains occurred repeatedly both day and night and were aggravated by using the leg. It had also been noted that lately there had been periods of infrequent urination. This was the first evidence of any involvement of bowel or bladder.

Re-examination. Because of this new development the child was seen in the out-patient department on January 20, 1934. His weight was 10.3 kilograms (22 pounds, 10 ounces). The lungs, heart, and abdomen seemed perfectly normal. The left leg seemed stronger than it had been. The knee jerks were both absent as was the ankle jerk on the left. No sensory loss could be detected. During the examination the patient suddenly cried out with severe pain in the left ankle. This persisted for about 10 minutes. During this time mobility of the leg remained unchanged. Subsequently the left lower extremity was "cold and clammy," while the right remained warm and dry. The mother stated that she had repeatedly observed the same change following similar attacks of pain.

Roentgenograms were made of the hips and pelvis but these were reported as normal.

The child was again returned to his home. On February 19, 1934, Dr. Carpenter wrote that the child seemed more emaciated than ever and was suffering greatly from the attacks of pain.

Readmission. On March 19, the child was readmitted to the clinics. The parents stated that the pains had gradually grown more severe until February 20, when the child had suffered a particularly bad attack. Following that a rash had appeared on the leg which according to the family physician followed "the nerve." The rash soon disappeared and subsequently he was practically free from pain, but developed a swelling on the external margin of the left foot.

His weight at this time was 9.6 kilograms (21 pounds, 2 ounces). The general physical examination except for the emaciation was negative. Neurological examination revealed weakness of both lower extremities, especially the left, absence of all tendon reflexes in the legs, wasting of the entire left lower extremity, rigidity of the lower back, and no sensory disturbance.

On March 26 a lumbar puncture was performed. The spinal fluid was deep amber in color and coagulated on standing.

Roentgenograms were made of the vertebral column from the seventh cervical vertebræ to the coccyx and were reported as normal.

On April 4, under ether anæsthesia, the cisterna magna was punctured and 1 cubic centimeter of lipiodol injected. Subsequent roentgenograms revealed that the lipiodol had descended the spinal canal to the level of the eleventh thoracic vertebra where it was completely arrested by a dome shaped mass (Figs. 1 and 2). With this additional information a review of this and previous roentgenograms revealed a gross dilatation of the spinal canal from the twelfth thoracic to the third lumbar vertebræ inclusive, in both the lateral and anteroposterior diameters. There was visible a distinct flattening of the medial surfaces of the pedicles of these vertebræ with definite narrowing of their transverse diameters. In the lateral views it was obvious that the pedicles, particularly those of the first lumbar vertebra were markedly decalcified.

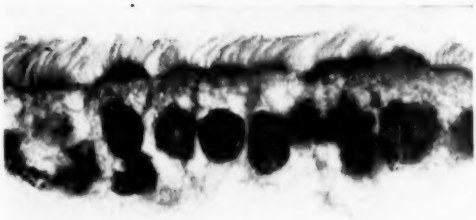

Fig. 5. Epithelial lining of the thin cyst wall. It is composed of a single layer of ciliated cuboidal epithelium. Hæmatoxylin and eosin. × 1310.

The child was again examined on April 9, at which time the upper extremities were normal. There was marked rigidity of the neck and entire back. The spinous processes of the ninth thoracic vertebra seemed unduly prominent but because of the severe emaciation all of the spines were very distinct. There was definite tenderness of the lower thoracic and lumbar regions posteriorly. The abdomen was somewhat distended but no localized weakness could be detected. All abdominal reflexes were active. The cremasteric reflex was present on the right but absent on the left. There was definite atrophy and weakness of both lower extremities. All movements were possible but weak. The patient was unable to stand even when grasping the sides of his bed but could sit up unaided. The right knee jerk was fairly active but the left was absent. Both ankle jerks were present and ankle clonus was elicitable on the right. Babinski's sign was positive bilaterally. There was definite flaccidity, especially of the left lower extremity. No sensory loss to pin prick, cotton, vibration or sense of position could be detected. Marked persistent erection of the penis was present throughout the examination and was repeatedly observed thereafter.

It seemed definite from this examination that the lesion of the nervous system which was previously manifest by weakness, atrophy and loss of tendon reflexes had now extended to a higher level in the spinal cord as shown by the hyperactive reflexes, clonus, and the presence of Babinski's sign.

Diagnosis. With the aid of the lumbar puncture and X-ray findings a diagnosis of a large tumor of the lower spinal cord and cauda equina was made.

Operation. April 10, 1934. Under ether anæsthesia the spines and laminæ of the eleventh and twelfth thoracic and upper four lumbar vertebræ were exposed and resected. The laminæ of the twelfth thoracic and upper three lumbar vertebræ were extremely thin and had been pushed far posteriorly, greatly reducing the length of the spinous processes. The spinal canal was dilated to two or three times the normal size (Figs. 3 and 4). The dura mater was thin and translucent. Within it was found a large thin walled cyst which lay beneath the arachnoid (Fig. 3). This cyst extended from the eleventh thoracic to the upper border of the fourth lumbar vertebra. It was filled with a very thick tenacious, glary, opalescent material which jelled when exposed to the air. The cyst was slowly dissected from the spinal roots which lay on either side and the spinal cord which was beneath, i.e. anterior to it. It was impossible to remove the cyst intact so the contents were evacuated (35 cubic centimeters, 38 grams) and the entire cyst removed. In the lower part at the level of the third lumbar vertebra, there was an elongated bean-shaped solid nodule measuring about 1.5 centimeters by 0.5 centimeter in the anterior wall. This was the only point at which the tumor was attached to the surrounding structures. Here entered all of its vascular supply. This nodule was firmly attached to the posterior surface of the spinal cord about 1.5 centimeters above the tip of the conus medullaris. It was carefully removed from the spinal cord by blunt dissection.

Fig. 6. Section through the entire thickness of the teratomatous mural nodule. *A*, Stratified ciliated columnar epithelium lining the cystic cavity; *B*, mucous glands; *C*, adipose tissue; *D*, hyaline cartilage; *E*, posterior columns of the spinal cord. Hæmatoxylin and eosin. × 15.

After the tumor was removed (Fig. 4) one could see the roots of the cauda equina lying against the wall of the dilated spinal canal and the spinal cord which had been flattened to a thin ribbon lying on the anterior wall of the canal. The tip of the conus medullaris extended almost to the lower border of the fourth lumbar vertebra. In the midline of the posterior surface of the lower end of the spinal cord was a defect from which the tumor had been dissected. The wound was closed without drainage.

Postoperative course. Following the operation the patient was incontinent but his recovery was otherwise uneventful. He was discharged on April 24, 1934, 14 days after operation. At that time examination revealed good strength in all muscles of the lower extremities except for those concerned with movement of the left foot, in which strength was only fair. Reflexes were normal except for increased ankle jerks with ankle clonus bilaterally and a positive Babinski's sign on the right. There was no sensory loss. The child was able to stand when grasping the sides of his crib-bed. Two weeks later, on May 8, the child was seen in the out patient department. The change in his condition was striking. His parents stated that whereas before they had had great trouble getting the child to eat, each meal requiring about 2 hours, he now ate readily and demanded more as well as having to be fed between meals. He no longer suffered from any pain. He slept very well. The child's face was fuller, and his color good in contrast to his pale, pinched expression before operation. He was able to

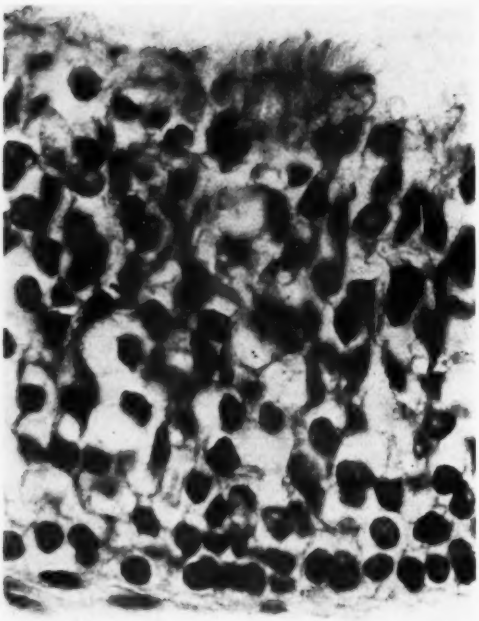

Fig. 7. Stratified ciliated columnar epithelium which covered the inner surface of the mural nodule. There are many large goblet cells. Hæmatoxylin and eosin. × 800.

stand without aid and to walk with very little assistance. There was no longer any trouble with either bowel or bladder. When last seen on June 5, 1934, the patient was able to walk quite well without any assistance.

Microscopical examination[1]. The wall of the cyst and the mural nodule were fixed in formalin, sectioned serially, stained with hæmatoxylin and eosin and impregnated by Freeman's silver technique for nerve fibers and Perdrau's method for reticulin.

The cyst wall consisted of two parts, a loose vascular connective tissue which covered its surface and a lining epithelium. In many places this epithelium was composed of a single layer of low cuboidal epithelium (Fig. 5). Elsewhere it was two to three cells in thickness. Upon the inner surface of these cells were many cilia.

The mural nodule was much more complex (Fig. 6). It consisted of two portions between which there was no sharp line of cleavage. That portion of the nodule nearest the cyst cavity was composed of teratomatous tissue. On its surface was a thick layer of ciliated stratified columnar epithelium in which many goblet cells could be seen (Fig. 7). Lying beneath this epithelium was a loose connective tissue in which were embedded many pieces of hyaline cartilage. Lying in close relation to the cartilage in a manner strongly suggestive of the trachea were found nests of glands (Fig. 8). It was the opinion of Dr. R. R. Bensley of the Department of Anatomy that these glands were mucous in type and most closely related to those normally found in the posterior part of the palate, although they bore some

[1] We are deeply indebted to Dr. George Bartelmez of our Department of Anatomy who very kindly assisted us in the microscopical examination of this tumor.

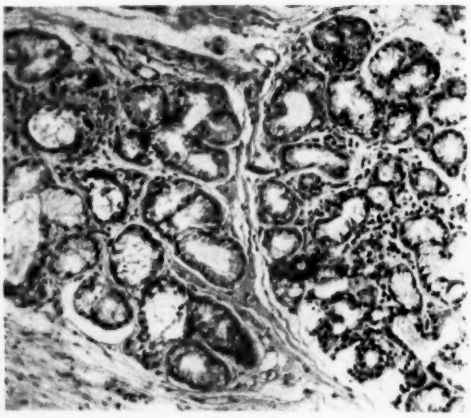

Fig. 8. Mucous glands from the mural nodule. Hæmatoxylin and eosin. × 102.

resemblance to those of the trachea. These glands were connected by several ducts to the interior of the cystic cavity into which they presumably poured their secretion. In this same area were found bundles of unmyelinated nerve fibers (Fig. 9). Through the serial sections these could be traced to their origin from a small group of large multipolar ganglion cells at one end of the mural nodule (Fig. 10). There were also areas of adipose tissue (Figs. 6 and 9) and several isolated collections of lymphocytes. No tissue which could be definitely identified as muscle could be seen.

That portion of the nodule farthest from the cystic cavity and in close contact with the spinal cord consisted primarily of a great number of longitudinally arranged, parallel nerve fibers which were best shown by Freeman's technique of silver impregnation for nerve fibers (Fig. 11). In places these fibers were closely crowded together, in others they were more sparse. Not all of them were normal, many being swollen and fragmented, while others possessed degenerative end-bulbs and end-loops. In places where the nerve fibers were few there was a proliferation of glial tissue. It was felt that most probably this area was a portion of the posterior columns of the spinal cord which was intimately connected with the teratoma and had been removed with it. This was regarded as particularly likely because of our inability to demonstrate any neurocytes from which these nerve fibers might have arisen. Those few ganglion cells which had been found were definitely related to the bundles of unmyelinated nerve fibers found in the more superficial parts of the tumor. At no point was there any evidence of any connection or relationship of the tumor or the cyst to the ependyma-lined central canal of the spinal cord.

Portions of the gelatinous contents of the cyst were fixed in various fixatives, embedded, and stained with hæmatoxylin and eosin. Microscopical examination revealed no definite cells. There were a number of large indistinct eosinophilic cytoid bodies. These were entirely devoid of nuclei. The gelatinous material itself presented a uniformly finely granular eosinophilic appearance.

NATURE AND GENESIS OF THE TUMOR

This tumor undoubtedly comes under the classification of teratoid or bidermal tumors. As Hos

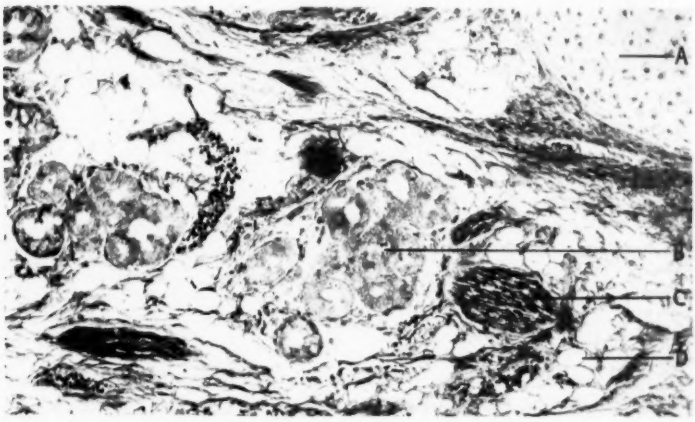

Fig. 9. Section from the mural nodule showing bundles of unmyelinated nerve fibers, *C;* in the neighborhood of the, *A*. hyaline cartilage; *B*, mucous glands; and *D*, adipose tissue. Freeman's method for nerve fibers. × 115.

pointed out, this is true for all examples of intradural teratoma. And as has been true in all other instances, the germinal layer not represented in the various structures which compose this tumor is the entoderm. All the structures present would be classified as arising either from ectoderm (ciliated stratified columnar epithelium, ciliated cuboidal epithelium, mucous glands, ganglion cells, unmyelinated nerves) or mesoderm (cartilage, adipose tissue, connective tissue). Such teratomatous neoplasms as this are probably not to be explained by a simple mechanical embryological malformation such as gives rise to dermoid and epidermoid cysts, or the congenital dermal sinuses recently reported by Walker and Bucy. In these, simple inclusion of a portion of the surface ectoderm in the underlying mesodermal tissues gives rise to a neoplasm or malformation composed solely of proliferated epidermis and dermal structures. This complex teratomatous neoplasm composed of elements largely devoid of any embryological relationship to the nervous system or its anlagen probably arose from a maldevelopment of the ovum, the misplacement of some multipotential germinal cells early in embryonic development, forming as Bucy and Haymond have pointed out for another similar tumor "a twin which had not gone on to full development." It would seem that the explanation most favored by Kubie and Fulton that these "cysts . . . probably represent ependymal diverticula of the central canal of the spinal cord" is an entirely inadequate explanation because of the lack of demonstrable relationship between the ependyma and such structures as cartilage, fat tissue, and mucous glands.

DISCUSSION OF THE CLINICAL ASPECTS

This case again demonstrates the value of lumbar puncture in cases of obscure diseases of the spinal cord. It is also illustrative of the point that the diagnosis of anterior poliomyelitis is particularly to be questioned when the manifestations are somewhat unusual or the disease is progressive. In the present case suspicion that the diagnosis which had been made was incorrect was first raised with the appearance of subjective pain. The pain can in all probability be attributed to irritation of the posterior lumbar nerve roots, although the possibility that it was secondary to intense vascular spasm must be considered; especially so as the attacks of pain were followed by a localized vasoconstriction which was manifested by the left lower extremity becoming "cold and clammy." These attacks may have been due to irritation of the vasoconstrictor nerve fibers arising from the spinal cord. However, it is generally conceded that the efferent nerves to the sympathetics arise only from the thoracic spinal cord whereas the tumor in this case was limited largely, if not entirely, to the lumbosacral portion of the cord. It is true that Oughterson, Harvey, and Richter have presented evidence of some vasoconstrictor fibers arising from the lumbosacral cord but it is questionable if irritation of these few fibers alone would have given rise to such severe vascular spasms. The attacks, of course, may have been elicited in some obscure reflex manner. The relationship of the pain and the vasoconstriction to the rash which subsequently developed is also not clear. This rash was presumably of the nature of a herpes zoster. And although the pathogenesis of

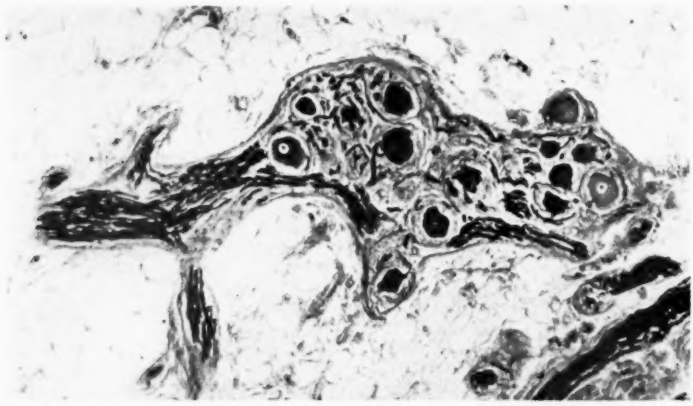

Fig. 10. Ganglion cells found at one end of the teratoma. The unmyelinated nerve fibers which pass throughout the tumor can be seen arising from the ganglion cells. At this point the ganglion cells and nerve fibers are embedded in adipose tissue. Freeman's method. × 130.

it be undetermined it is of interest to know that intraspinal neoplasms can produce such a condition.

With the appearance of these phenomena obviously not explicable on the basis of an anterior poliomyelitis, the progression of the paralysis and the development of hyperactive reflexes, clonus, and positive Babinski's sign, it was evident that the previous diagnosis was incorrect.

The lumbar puncture which revealed the typical findings of a complete obstruction of the spinal subarachnoidal space confirmed this belief.

The roentgenograms (Figs. 1 and 2) of the lumbar spine were typical of an enormous expanding intraspinal lesion. Similar pictures have been observed in other cases in the cervical as well as the lumbar region. One such case with a very large ependymoma filling the entire lumbar canal dilating it and eroding the pedicles and laminæ has been recently reported from this clinic by Hamby.

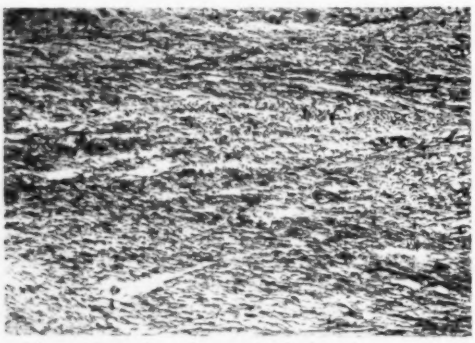

Fig. 11. Posterior columns of spinal cord removed with mural nodule. This area is composed of a large number of nerve fibers all running in the same axis, parallel to the spinal cord. Many fibers are degenerated and close inspection will reveal swollen fibers and a few degenerative end-bulbs even at this low magnification. Freeman's method. × 70.

Lipiodol was utilized in this case because with the absence of any sensory change, the confusing nature of the motor alteration, both flaccid and spastic phenomena being present, it was impossible from the neurological examination accurately to determine the upper limit of the tumor. As is so often true with tumors in this region (Harkins) the illustrations show that the lipiodol successfully defined the upper limit of the neoplasm. In this clinic no irritation of more than very temporary duration and slight intensity has ever been observed from lipiodol introduced into the subarachnoidal space, and there would seem no reason for hesitation in its employment whenever it is indicated.

It has been apparent in many cases that severe compression of the spinal cord may be compatible with retention of normal or nearly normal sensation. This seems especially true in children and has been noted by Walker and Bucy in cases of subdural abscess of the spinal cord in which there was marked alteration in the motor functions of the spinal cord with but little effect upon sensation. Why this should be so is not apparent. It is true that in the present instance the paralysis was probably in a large measure the result of compression of the anterior horn cells rather than the pyramidal tract. However, that is a situation

TABLE I.—REPORTED CASES OF TERATOID TUMORS OF THE SPINAL CORD

Author	Sex, age in years	Location	Size	Ectodermal structures	Mesodermal structures	Structures hard to classify
1. Gowers, 1876	.. Adult	Conus medullaris	0.5 x 0.5 x 0.4 in.		Connective tissue, striated muscle, fat	
2. Gerlach, 1894	M 36	Upper cervical	about 3 mm. long		Connective tissue, primitive and adult muscle fibers, cartilage	Embryonal tissue
3. Forbes, 1905	M 5.5	Midcervical	Size of haricot bean		Cellular connective tissue, embryonal and adult striated muscles, fat cells, multinucleated giant cells (osteoclasts?)	
4. Frick, 1911	F 41	Second to fifth lumbar vertebræ		Numerous blond hairs	Connective tissue, bone with myeloid tissue in marrow, fat	
5. Andre-Thomas and Quercy, 1912	..	Cervical and dorsal cord		Ependymal cells, glial elements (glioma)	Connective tissue, striated muscle, numerous blood vessels	
6. Bielschowsky, and Unger, 1920	M 37	Third cervical	Twice walnut size	Fine hairs	Connective tissue, fat cells, endothelioma or alveolar sarcoma	
7. Hansmann, 1926	F 21 days	Sacral	6 cm. diameter	Stratified squamous epithelium, nerve, glial cells, ependyma, ependymal glioma (?).	Myxomatous connective tissue, smooth muscle, cartilage, fat, lymphoid cells	Cysts lined with high columnar cells
8. Kubie and Fulton, 1928	M 2	9th dorsal to 1st lumbar	6 x 1.5 cm.	Mucous and serous glands, large myelinated nerve fibers	Connective tissue, smooth muscle	Cysts lined with ciliated columnar cells
9. Kubie and Fulton, 1928	F 27	3rd and 4th cervical		Mucous and serous glands, myelinated nerve fibers, ganglion cells (?)	Connective tissue, smooth muscle, fat, cartilage, lymphoid tissue	Cysts lined with ciliated cuboidal to columnar cells
10. Hosoi, 1930	M 24	2nd and 3rd lumbar	About 1.5 x 3.5 cm.	Nerve fibers, pacinian corpuscles	Connective tissue, smooth muscle, fat cells	Glands lined with ciliated and nonciliated columnar cells
11. Bucy and Buchanan, 1934	M 2¾	11th dorsal to 4th lumbar		Ciliated stratified columnar epithelium, ciliated cuboidal epithelium, mucous glands, unmyelinated nerve fibers, ganglion cells	Connective and adipose tissues, blood vessels, cartilage, lymphoid tissue	

peculiar to the present case whereas the fact that the sensory pathways are more resistant to pressure than the motor is borne out by cases of tumors in all parts of the spinal cord. In fact this observation concerning the greater resistance of the sensory pathways is not limited to such agents as mechanical pressure but is also seen in other conditions. For instance in both multiple sclerosis and progressive muscular atrophy sensory changes are uncommon and of little moment, whereas microscopical examination of the spinal cords from such cases often reveals marked demyelinization which is not limited to the efferent or motor tracts but is disseminated throughout the spinal cord.

One is also often amazed at the amount of recovery which occurs after such a severe degree of spinal cord compression. This seems especially true of children and was observed in the cases of subdural abscess as well as in the present case, in all of which the compression was of long standing. Not only is the degree of recovery striking but its rapidity as well. In the present instance the patient after months of illness, malnutrition, and incapacity was able to stand 2 weeks after operation and to walk in 4 weeks.

LITERATURE

The reports dealing with intradural teratomata were thoroughly dealt with by Hosoi in 1931. There do not appear to have been any further additions to the literature since that time. It does not seem necessary to repeat Hosoi's review, rather I have appended his table (Table 1) to which I have added the present case.

In 1932 Bucy and Haymond recorded an example of a teratoma arising outside of the spinal canal but connected with its neural contents by a narrow stalk. They found two similar cases, those of Sonntag and Aloi, in the literature. These extraspinal teratomata, all intimately connected with the nervous system, are in all probability closely related to those arising within the dura. They are very similar in gross and microscopical appearance.

SUMMARY

A case of bidermal teratomatous tumor arising from the posterior columns of the lower end of the

spinal cord is here recorded. The tumor lay within the arachnoid and was obviously of long standing, for it had produced a marked dilatation and erosion of the spinal canal. The growth had severely compressed the spinal cord, this compression in turn giving rise to a mixed and alternating spastic and flaccid paraplegia, with considerable subjective pain but without detectable sensory disturbance. A laminectomy was made, and the entire cystic neoplasm was removed. This operation was rapidly followed by a striking recovery of all functions.

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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JUNE, 1935

SYMPATHETIC OPHTHALMIA

IN the group of inflammatory diseases of the uveal tract which come under the general classification of uveitis, sympathetic ophthalmia stands out as a clinical entity. It follows penetrating injuries to the eye, operations on the eye involving the iris or ciliary body, and, rarely, non-penetrating injuries, such as contusion, and intra-ocular growths. The incidence is comparatively rare, yet the insidiousness of the disease and the great probability of total loss of vision when it occurs make it the most dreaded of complications in cases of injury to the eye. Early diagnosis, prophylaxis, and treatment are of great importance from the standpoint of industrial compensation.

The onset of symptoms has been noted 2 days after the injury; the average period, however, has been from 2 weeks to 6 weeks; the longest period reported has been 48 years. The symptoms of sympathetic ophthalmia are not always easily recognizable in the early stages, particularly in cases in which the onset is delayed or in which there is obscure or uncertain evidence of initial injury, but by biomicroscopic examination of the aqueous humor in the post-lental space and about the inferior angle of the anterior chamber, the presence of cells may be detected before other signs of the disease appear. Low intra-ocular tension, mild circumcorneal injection, cells appearing in the aqueous humor of a previously healthy eye a few days or weeks following a penetrating injury to the other eye, constitute sufficient grounds for presumptive diagnosis of sympathetic ophthalmia and warrant energetic therapeutic measures.

The answer to the question of what therapeutic measures are adequate to arrest the disease is not to be found in any textbook or treatise. As is usually the case when an adequate method of treatment is not known, many methods are recommended. A basis for specific treatment depends on etiology. There are many theoretic and some demonstrable explanations for development of sympathetic ophthalmia, none of which is conclusive, but the most highly regarded theories at present are as follows: (a) infection from without by a bacterial agent that has, or that acquires, affinity for uveal tissue by penetration into the injured eye, whereupon it becomes particularly potent and affects the fellow eye by transmission through the blood or lymph or by migration along the optic nerves; (b) the presence of a filtrable virus somewhat similar in action to the virus of herpes; (c) allergy, particularly to uveal pigment; and (d) endogenous tuberculosis. There are some reasons for believing that the etiological factors may be different in different cases, but the histopathological picture is characteristic and does not support this assumption. It is ex-

tremely difficult to reproduce the disease experimentally. Although a great deal of information has been gained from laboratory studies of the disease, treatment must yet be based on clinical experience.

During the decade of the World War there was developed a great confidence in the efficacy of parenteral injection of foreign proteins as a prophylactic against, and for treatment of, ocular inflammation following injury. The value of antitetanic serum after injury was generally acknowledged, but it was regarded as a specific agent. The low incidence of sympathetic ophthalmia from injuries to the eyes of soldiers in the war has been attributed to the use of antitetanic serum and prophylactic vaccines. Fear of anaphylaxis restricted its applicability in some cases. There were, however, no dangerous complications following parenteral injection of sterilized milk or properly-prepared typhoid vaccine. Diphtheria antitoxin was regarded as particularly dependable and safe for children, and as sympathetic ophthalmia occurs most often in children, the remedy became very popular.

When used as a prophylactic, foreign proteins are so effective in preventing the disease that sympathetic ophthalmia now is regarded as one of the most unlikely complications of surgery of the globe, or following injury to the eye, even when the operation, or the injury, is in the so called danger zone. By energetic treatment of the disease in the early stages by the same means, it often can be arrested and vision saved. This clinical experience points out two obvious and significant truths: (1) that effective therapeutic measures may be developed on the basis of clinical experience without regard to the results of laboratory investigation of etiological factors, and without a full understanding of the ways in which healing is brought about; and (2) that clinical experience in the use of foreign protein for prophylaxis and treatment of sympathetic ophthalmia has shown it to be so effective when properly given that it should be adopted as a routine procedure in every case of injury that makes the eye subject to the disease.

W. L. BENEDICT.

PNEUMONECTOMY AND LOBECTOMY

UNTIL 4 years ago no patient had ever survived the removal of an entire lung, and until 6 years ago the danger of the complete removal of a pulmonary lobe was so great as to be prohibitive. The phenomenally rapid technical advances that have been made in the operations of pneumonectomy and lobectomy during the last few years furnish another illustration of the successful application of physiological principles to surgical problems.

During the last 4 years at least six total pneumonectomies have been successfully performed for bronchiectasis involving an entire lung, and at least twelve pneumonectomies for malignant disease. Although the present mortality rate of approximately 25 per cent will probably be considerably reduced by the application of the lessons already learned, pneumonectomy will always be a relatively dangerous operation. A simple wide exploratory thoracotomy, undertaken to determine the operability of a carcinoma in an old person whose general resistance is poor is, however, apt to prove to be a more hazardous operation than the actual removal of a lung in a young or middle aged person in good condition.

The fact that pneumonectomy has been demonstrated to be possible, and not particularly hazardous in patients in reasonably good condition, has already stimulated interest among internists and bronchoscopists in the diagnosis of bronchial carcinoma in the operable stage. As a direct result of this

growing interest in early diagnosis, it is almost certain that a steadily increasing number of successful pneumonectomies, followed by indefinitely prolonged freedom from recurrence of carcinoma, will be reported.

The surgical problems presented by carcinomatous and bronchiectatic patients are quite different. Bronchial carcinoma is frequently located so centrally that the stem bronchus must be divided in its intramediastinal portion after the separate ligation of the pulmonary artery and veins. The dissection within the mediastinum and the healing of the sutured bronchial stump are favored by the one stage operation that is usually possible in cases of carcinoma. A one stage operation is feasible because the absence of pronounced pulmonary suppuration in the operable phase of carcinoma reduces the danger of infection of the pleural cavity, and the absence of extensive pleural adhesions reduces the operating time. The pleural cavity may be tightly closed without tube drainage, thus permitting the postoperative pleural exudate to become rapidly organized and pull the ribs, hemidiaphragm, mediastinum, and remaining lung toward the pleural cavity until it is obliterated. In this event a thoracoplasty is, of course, unnecessary.

All the successful pneumonectomies for carcinoma have been one stage operations, and for bronchiectasis two stage. A one stage operation in a bronchiectatic patient would be unduly hazardous because virulent infection would probably attack the pleura that has not been protected by the traumatic inflammatory wall that a preliminary stage would have produced. Open drainage of the empyema, or the early opening of the infected hilar bronchus, after a one stage operation would expose the patient to grave danger of cardiorespiratory decompensation from pendulum respiratory movements of a mediastinum that was as yet mobile. Since the stem bronchus need not be divided within the mediastinum in bronchiectatic patients, the extramediastinal portion of the hilum, or preferably the individual lobar hila, may at the second stage be circumferentially ligated, or sutured after their division. Because of the extensive pleural adhesions that are usually present in patients having bronchiectasis of an entire lung, a posterior incision is best, whereas an anterior incision is preferable for carcinoma patients because it gives direct access for the dissection of the pulmonary artery and veins in the anterior part of the hilum, and because pleural adhesions are usually only few in number.

The mortality rate from the newly developed lobectomy operations for unilateral bronchiectasis (in bilateral disease the rate is prohibitively high) has been reduced to less than 13 per cent in a total of 116 patients operated on in the four clinics in which the death rate is the lowest. Fifty-nine of these patients were operated on by the two stage technique with a mortality rate of 10.2 per cent, and the remaining 57 by the one stage technique with a mortality rate of 12.5 per cent. The published mortality rates in other clinics using the one stage operation are 11.1 per cent, 23 per cent, 25 per cent, and 29.6 per cent, respectively. In spite of the lower death rate of the two stage operation, there is at present a sharp division of opinion as to which operation is the better.

In the two stage lobectomy, the undiseased lobe is adherent at the time that the second stage is performed, and the mediastinal, diaphragmatic, and costal pleura over the lobe that is to be removed contains a traumatic inflammatory barrier against the infection that will follow the removal of the lobe. These two factors protect the patient against the development of a virulent total empyema, and

against the pendulum respiratory movements of the mediastinum and the paradoxical movements of the ipsilateral lobe that would probably occur if the hilar bronchus opened prematurely, or if the thoracic wall incision could not be kept tightly closed during the first week or two after operation. The chief disadvantages of the two stage operation are that the patient must be operated on twice, and that the empyema that occupies the space that contained the diseased lobe is larger and takes longer to become closed than does the empyema remaining after the one stage operation, for the reason that the adhesion of the remaining lobe to the thoracic wall prevents its expanding rapidly to fill the empyema cavity. A further disadvantage of the two stage technique is that approximately half of the patients require a muscle graft operation to close a residual bronchial fistula.

The one stage operation is convenient for the patient and surgeon and, by virtue of the rapid filling of the space from which the diseased lobe was removed, the postoperative convalescence is relatively short; the bronchopleural fistula that develops usually closes spontaneously. The chief disadvantages of the one stage operation are: (1) the relatively long operating time required increases the danger of shock and postoperative pneumonia; (2) the tourniquet that is used to control the hilar vessels while the hilum is being closed by suture may slip with a resulting fatal hæmorrhage; (3) some of the many divided hilar vessels cannot be identified for certain closure by the hilar sutures, and may be the cause of a fatal postoperative hæmorrhage (in the two stage operation the hilum is usually tightly ligated *en masse*, and operative or postoperative hæmorrhage is a rare complication); (4) severe infection of part or all of the pleural cavity sometimes occurs. In spite of certain obvious advantages of a one stage lobectomy, the two stage procedure seems at the present time to be the safer and, therefore, the preferable operation.

The progress that has already been made with the operations of pneumonectomy and lobectomy justifies the inclusion of pulmonary carcinoma and intractable cases of unilateral bronchiectasis among the curable diseases.

JOHN ALEXANDER

EARLY AMERICAN MEDICAL SCHOOLS

THE MEDICAL FACULTY OF LAVAL UNIVERSITY, QUEBEC

PROFESSOR A. VALLÉE, M.D., F.R.C.P.C., QUEBEC, CANADA

THE Medical Faculty of Laval University has been teaching for some 80 years, since it was organized in the fall of 1853. To understand its real origin, one must go back as far as the beginning of the French colony of Nouvelle France, and follow up the evolution of French teaching which has been followed since the foundation of the school.

In 1663, the first Bishop in North America, François de Montmorency Laval, established the Quebec Seminary. In 1676, a Royal Charter was received from the French King, Louis the XIV. The Seminary created Laval University in 1852 and obtained then a Royal Charter from Queen Victoria of England together with the Papal Bullas as a Catholic university. From this moment Laval jealously preserved that classical education of the French and other great universities in Europe, such as Oxford, Cambridge, Rome, and Louvain. Laval has four faculties: divinity, law, medicine, and arts comprising sciences and letters. French is the teaching language, and technique books in the medical faculty are all in French.

These notions were necessary to have in full sight the situation of the Medical Faculty of Laval University.

Before its foundation, the student who desired to enter medical practice in Quebec City, had to study with a practitioner who could initiate the future physician to the first notions of medical science and the practice of medicine and surgery. Since 1847 a medical school had been organized which was called: L'École de Médecine incorporée de la Cité de Québec. In this school medicine and surgery were taught and clinical lectures were given at the Marine Hospital, while dissecting rooms were provided successively in different parts of the city. The professors of this school were called upon to become the first professors of the Medical Faculty of Laval. At the opening of the university in 1854, the faculty had 6 professors with Dr. Jean Blanchet as dean. It counts actually 20 professors, 5 are full time; 19 associate professors; 6 in charge of lectures, and 27 assistants. Of this staff of 66, 51 have had postgraduate work in Paris, Strasbourg, Bordeaux, Lyons, for periods varying from 1 to 3 years; others have done postgraduate work in New York or other American cities.

To be admitted to the medical faculty, the student must either be a bachelor of arts of Laval or any recognized university, or have passed the special examination of admission to the study of medicine in the Province of Quebec established by the Medical Board of the Province, or submit the regulations of admission wanted in the Province or country to which he belongs. For American students, they must prove to have had 3 years of premedical work for entrance in the first year, and have been studying in a medical faculty classified "A" for other years. The students numbered 263 in 1933–1934.

The course in medicine is a full 5 year medical course of 8 months. All lectures are given in French. Semestrial examinations take place at the end of January and the beginning of May. Final examinations start on the fifteenth of May in every year. The presence of every student to lectures, practical work, and clinics is controlled every day. Lectures and practical work take place in the building of the medical faculty, which was rebuilt in 1922; clinics are given in the different hospitals connected with the faculty which include: The Hôtel-Dieu de Quebec, St-Sacrement Hospital, Laval Hospital, Maternity Hospital, Infant's Hospital (Crèche St-Vincent de Paul), Neuropsychiatric Clinic (Clinique Roy-Rousseau) and Insane Asylum (Hôpital St-Michel-Archange). Nominations in these hospitals are all controlled by Laval University, the total beds numbering over 4,000 of which 2,000 belong to the Insane Asylum.

Technical books on every subject are indicated to the students and must be bought at the university at the beginning of each year without the possibility of handling them over. The rest of the material, comprising microscopes and experimental apparatus, microscopic and macroscopic collections of specimens, is furnished without charge in all departments.

A library of 5,000 books, with 145 periodicals, is at the disposal of the students at the medical faculty in addition to the possible use of the university library which contains over 150,000 volumes. The library is opened daily from 9:00 a.m. to 6:00 p.m. and from 8:00 to 10:00 in the evening. Special indexes are at the disposal of the workers.

Special lectures are given every year by noted medical men and students are admitted to all meetings of the medical societies at the school or in the hospitals.

MEDICAL SCHOOL

In 1922 owing to the activities of a group of professors and the elaborate direction given by the new

dean, Professor A. Rousseau, the school has been totally rebuilt and all departments organized to look after the practical training of an average of 50 to 60 students per year.

On the top floor stands the department of biology, histology, and embryology under the direction of Professor Potvin, with large practical work laboratory for the first-year students with a possibility of accommodation for over 80 students, private laboratory for the professors and helpers.

Next is the department of anatomy directed by Professor Garneau. The large dissecting room as well as the annexed lecture room can be attended by 100 workers. Complete collections of bones, normal anatomy specimens, charts and moldings are at the disposal of the students and large working shops are fitted to prepare all the material at the school. Private laboratory for the professor and for the prosectors with all necessary outfits completes the material organization for anatomy teaching.

The next floor is entirely occupied by the services of physiology and biological chemistry, comprising laboratories for practical work in physiology at which students are trained by groups, large laboratory for biological chemistry, laboratories and offices for the professors and work rooms to prepare the material together with a large lecture room. The stables of the school close to the building are under the direction of this department together with the department of biology and pathology. Physiology is under the direction of Professor Blanchet and biological chemistry actually in charge of Dr. Marcoux.

The second floor of the school is occupied by the department of pathology and bacteriology. The Pathological Institute directed by Professor Vallée and Berger with the assistance of Dr. E. Morin, associate professor, examined and made report last year on 3,228 microscopic specimens. This department looks after the Cancer Center of Laval University which is directed by the same staff together with Professor Vezina as surgeon. The department is fitted with a large laboratory for practical work and 5 private laboratories for the staff. The Museum of Pathology contains over 500 macroscopic specimens of very first quality. A pathological collection of over 12,000 microscopic specimens is at the disposal of students who receive free of charge a complete series of all lesions amounting to 115 slides. Fourteen papers were published by the staff during the last year and over 50 since the foundation of the Institute in 1928. Charts, microscopic drawings, and microscopic photographs are prepared in the department.

General lecture rooms, registrar's offices, reading room, and library occupy the main floor together with the dean's office and professors' meeting room. In the basement are the vestries, recreation room, experimental hospital for animals, book room of the library, and the storing department for anatomy.

UNIVERSITY HOSPITALS

Students are obliged to follow hospitals during the third, fourth, and fifth year, but are admitted even during the first 2 years. They follow the different services by rotation either as *stagiaires*, or as externes for those who have had over 75 per cent on their marks on all subjects during the first and second year or as internes in the fifth year for those who after externeship have obtained 75 per cent on all examinations during the third or fourth years.

Owing to the large number of beds provided by the different university hospitals as mentioned, students are distributed in very small groups in each of the medical, surgical wards or in special services and maternity. They have to look after a certain number of patients of which they are in charge, under control of the assistants and house surgeons and physicians, to report cases and besides attend the didactic clinical lectures which take place in every hospital. *Stagiaire* students rotate every 3 months, externes every 6 months, maintaining their service during the whole summer months as do the internes.

Diplomas are delivered after the fifth year to all who went through with success all written, oral, and clinical examinations, in medicine, surgery, midwifery, and pediatrics for the clinical part. No student can change from one year to another if he has failed on any subject.

The dean of the Faculty of the school is actually Professor P. C. Dagneau, and the secretary is Professor A. Vallée.

CORRESPONDENCE

MISSISSIPPI VALLEY MEDICAL SOCIETY

To the Editor: A new medical organization to be known as the Mississippi Valley Medical Society was formally organized at Quincy, Illinois, on April 8. The sole purpose of the new society is to hold an annual meeting each fall devoted to intensive postgraduate instruction and conducted by the leading clinical teachers of the United States. The programs will be eminently practical and of particular interest to the general practitioner. The first meeting will be held in Quincy during the month of October or November and will be a 3 day session.

The control of the organization is in the hands of a board of directors, consisting of one director to each 1000 physicians in the states of Illinois, Missouri, and Iowa. Membership in the society will be open to all ethical physicians, it being a prerequisite that all members hold membership in their respective state medical societies. In order to get started quickly, the board of directors has elected to place the membership fee and dues for the first year at only $3.00 and life membership at $25.00, provided these are paid before the time of the annual meeting. Charter membership will close July 1, 1935. Members will attend the annual meeting without payment of a registration fee.

Ethical physicians interested in the new organization are urged to communicate with Harold Swanberg, M.D., secretary-treasurer, 211–224 W. C. U. Bldg., Quincy, Illinois.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

PRATIQUE ANATOMO-CHIRURGICALE ILLUSTRÉE. By F. Paitre, D. Giraud, and S. Dupret. ABDOMEN. FASCICULE II. RÉGION ABDOMINALE MOYENNE ET RECTUM. Paris: G. Doin & Cie, 1935.

SURGICAL PATHOLOGY OF THE PERITONEUM. By Arthur E. Hertzler, M.D. Philadelphia, Montreal, and London: J. B. Lippincott Co., 1935.

LA ROENTGENTHÉRAPIE DES FIBROMYOMES DE L'UTÉRUS ET DES MÉTROPATHIES HÉMORRAGIEUES. By Paul Gibert. Paris: Masson et Cie, 1935.

SEGUNDO CONGRESO ARGENTINO DE OBSTETRICIA Y GINECOLOGIA, Buenos Aires, 1934.

ECONOMIC PROBLEMS OF MEDICINE. By A. C. Christie, M.S., M.D. New York: The Macmillan Co., 1935.

THE PRINCIPLES AND PRACTICE OF UROLOGY. By Frank Hinman. Philadelphia and London: W. B. Saunders Co., 1935.

LE FONCTIONNEMENT DE L'ESTOMAC APRÈS GASTRECTOMIE. By Charles Marx. Paris: Louis Arnette, 1935.

AIDS TO SURGERY. By Cecil A. Joll, M.S. (Lond.), M.D. (Bris.), B.Sc. (Lond.), F.R.C.S. (Eng.), and Reginald C. B. Ledlie, M.B., B.S. (Lond.), F.R.C.S. (Eng.). 6th ed. Baltimore: William Wood & Co., 1935.

CLINICAL LABORATORY METHODS AND DIAGNOSIS; A TEXTBOOK ON LABORATORY PROCEDURES WITH THEIR INTERPRETATION. By R. B. H. Gradwohl, M.D. St. Louis: The C. V. Mosby Co., 1935.

PHYSIOLOGY IN MODERN MEDICINE. By J. J. R. Macleod, M.B., LL.D., D.Sc., F.R.C.P., F.R.S. Assisted in the present edition by Philip Bard, Edward P. Carter, J. M. D. Olmsted, J. M. Peterson, N. B. Taylor. 7th ed. St. Louis: The C. V. Mosby Co., 1935.

PHYSICAL DIAGNOSIS. By Warren P. Elmer, B.S., M.D., and W. D. Rose, M.D. 7th ed. St. Louis: The C. V. Mosby Co., 1935.

METHODS OF TREATMENT. By Logan Clendening, M.D., 5th ed. St. Louis: The C. V. Mosby Co., 1935.

DISEASES OF THE SKIN. By Richard L. Sutton, M.D., Sc.D., LL.D., F.R.S. (Edin.), and Richard L. Sutton, Jr., A.M., M.D., L.R.C.P. (Edin.). 9th ed. rev. and enl. St. Louis: The C. V. Mosby Co., 1935.

PRAKTISCHE ANATOMIE; EIN LEHR- UND HILFSBUCH DER ANATOMISCHEN GRUNDLAGEN ÄRZTLICHEN HANDELNS. By Dr. T. von Lanz and Dr. W. Wachsmuth. Band I, Teil 3. ARM. Berlin: Julius Springer, 1935.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

ROBERT B. GREENOUGH, Boston, *President* DONALD C. BALFOUR, Rochester, *President-Elect*

HOWARD C. NAFFZIGER, *Chairman;* THOMAS F. MULLEN, *Secretary, Committee on Arrangements*

THE 1935 CLINICAL CONGRESS IN SAN FRANCISCO AND OAKLAND

THE twenty-fifth annual Clinical Congress of the American College of Surgeons will be held in San Francisco and Oakland, October 28–November 1, 1935. Under the leadership of a strong and representative committee, the surgeons of that great medical center on the Pacific coast have organized and are planning to provide a program of surgical clinics and demonstrations in their hospitals and medical schools that will present a complete showing of their clinical activities in all departments of surgery. The Committee on Arrangements has been assured of the hearty co-operation of the clinicians at the two medical schools and twenty-seven hospitals that will participate in the clinical program.

A preliminary program of clinics and demonstrations as prepared by the Committee on Arrangements is published in the following pages. It will be noted that operative clinics and demonstrations in the hospitals are scheduled for the afternoon of Monday, October 28, beginning at 2 o'clock, and for the mornings and afternoons of each of the four following days. The schedules published at this time in tentative form will be revised and amplified as the work of the program committee progresses.

Among the special features of this year's clinical program will be: (1) Cancer clinics demonstrating the treatment of cancer by surgery, radium and X-ray; (2) fracture clinics demonstrating modern methods of treatment; (3) clinics in traumatic surgery demonstrating the newer methods of rehabilitation of injured patients by surgery and physiotherapy.

In addition to the ophthalmological and otolaryngological clinics and demonstrations at the hospitals and medical schools, the committee in charge of the section on surgery of the eye, ear, nose and throat is preparing programs for scientific sessions on Tuesday, Wednesday, Thursday and Friday mornings at headquarters at which distinguished specialists in these branches of surgery will present and discuss papers on subjects of clinical interest.

The following hospitals and medical schools will participate in the clinical program:

San Francisco—Children's, Franklin, French, Letterman General, Mary's Help, Mount Zion, St. Francis, St. Joseph's, St. Luke's, St. Mary's, San Francisco, Shriners', Southern Pacific, Stanford University, United States Marine, University of California, Veterans Administration; Stanford University School of Medicine, University of California Medical School.

Alameda County—Alameda County, Alameda Sanatorium, Alta Bates, Berkeley General, Children's, Cowell Memorial, East Oakland, Peralta, Providence, Samuel Merritt.

The Executive Committee of the Board of Regents is preparing programs for a series of five evening sessions. At the presidential meeting in the Opera House on Monday evening, the retiring president, Dr. Robert B. Greenough, of Boston, will deliver the annual address, and the new officers will be inaugurated: Dr. Donald C. Balfour, Rochester, president; Dr. Arthur W. Allen, Boston, and Dr. John A. Gunn, Winnipeg, vice-presidents. The American College of Surgeons oration will be delivered by Dr. George Crile, of Cleveland. Sessions on Tuesday, Wednesday and Thursday evenings will be held in the Auditorium of the Veterans' Building when eminent surgeons of the United States and Canada, together with visiting surgeons from foreign countries, will present papers dealing with surgical subjects of timely importance. The annual convocation of the College will be held on Friday evening in the Opera House at which the 1935 class of initiates will be received into Fellowship in the College. The fellowship address will be delivered by Dr. Robert Gordon Sproul, president of the University of California.

Among the special features of the program for this year's Clinical Congress will be: (1) A symposium on cancer, under the auspices of the College Committee on the Treatment of Malignant Diseases, on Thursday afternoon following the annual meeting, at which further reports by clinicians from various parts of the country will present additional statistics on the cure of cancer. Other papers on the treatment of cancer descriptive of modern methods of treatment and the organization and administration of cancer clinics will be included. (2) A conference on fractures, arranged in co-operation with the College Committee on the Treatment of Fractures on Tuesday afternoon. (3) A conference under the auspices of the Board on Industrial Medicine and Traumatic Surgery on Friday afternoon. All of these sessions will be held in the Gold Ballroom of the Fairmont Hotel.

HOSPITAL CONFERENCE

The annual hospital conference will open the Congress with a session in the Gold Ballroom of the Fairmont Hotel at 10 o'clock on Monday morning. An interesting program of papers, round table conferences and practical demonstrations dealing with problems related to hospital efficiency is being prepared for sessions to be held on Monday, Tuesday, Wednesday and Thursday in the Gold Ballroom of the Fairmont Hotel, and at several of the hospitals. A greatly increased interest on the part of surgeons in both the administrative and scientific phases of hospital work has been evidenced in recent years and the program for this year's conference will be unique in providing for discussions of subjects of interest to the three major hospital groups—medical, surgical and administrative. It is planned to make this year's program of wide interest and practical character through a careful selection of subjects to be presented and discussed by surgeons and hospital executives, particular emphasis being directed toward professional standards and the vital problems related to medical economics.

HEADQUARTERS—TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Fairmont and Mark Hopkins hotels. At the former the Terrace Ballroom and Lounge, the Gold Ballroom and other large rooms on the main floor and on the terrace have been reserved for scientific sessions and conferences, registration and clinic ticket bureaus, bulletin boards, exhibits, executive offices, etc. The Peacock Court and Room of the Dons at the Mark Hopkins will be utilized for various scientific sessions.

The Technical Exhibition, including the registration and clinic ticket bureaus, will be located in the ballroom and lounge on the terrace floor of the Fairmont Hotel. In these rooms will also be found the bulletin boards on which the daily clinical program will be posted each afternoon. The leading manufacturers of surgical instruments, X-ray apparatus, operating room lights, hospital apparatus and supplies, ligatures, dressings, pharmaceuticals and publishers of medical books will be represented in this exhibition.

LOW RAILWAY FARES

Very low railway fares will be in effect for the Congress, the railroads of the United States and Canada having authorized an extension of selling dates and return limit for round-trip summer tourist tickets to include the dates of the Clinical Congress with a return limit of November 30.

SAN FRANCISCO HOTELS AND THEIR RATES

In addition to the two headquarters hotels—the Fairmont and Mark Hopkins—there are a number of first-class hotels within short walking distance of headquarters providing ample hotel facilities at reasonable rates. The following hotels are recommended by the Committee:

	Minimum Rate with Bath	
	Single	Double
Bellevue, Geary and Taylor	$3.00	$4.00
Californian, Taylor and O'Farrell	3.00	4.50
Clift, Geary and Taylor	3.50	5.00
El Cortez, Geary near Taylor	3.00	4.50
Fairmont, Mason and California	3.50	5.00
Gaylord, Jones near Geary	3.00	4.00
Mark Hopkins, Mason and California	3.50	5.00
Palace, Market and New Montgomery	3.50	5.00
Plaza, Post and Stockton	3.00	4.00
Sir Francis Drake, Powell and Sutter	3.50	5.00
Stewart, 353 Geary	2.50	4.00
St. Francis, Union Square	3.50	5.00

ADVANCE REGISTRATION

The hospitals and medical schools of San Francisco and Oakland afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to a number that can be comfortably accommodated at the clinics—the limit of attendance being based upon the result of a survey of the amphitheaters, operating rooms, and laboratories of the hospitals and medical schools to determine their capacity for visitors. It is expected, therefore, that those surgeons who wish to attend the Congress will register in advance.

Admittance to all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the

distribution of the visiting surgeons among the several clinics and insures against overcrowding, as the number of tickets issued for any clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.

COMMITTEE ON ARRANGEMENTS

EXECUTIVE COMMITTEE

HOWARD C. NAFFZIGER, Chairman; THOMAS F. MULLEN, Secretary

LEROY C. ABBOTT
HAROLD BRUNN
EDMUND BUTLER
WHITFIELD CRANE
CHARLES A. DUKES
LEO ELOESSER
PHILIP K. GILMAN
OLIVER D. HAMLIN
FRANK HINMAN
EMILE F. HOLMAN
FRANK W. LYNCH
ALSON R. KILGORE
JOSEPH L. MCCOOL
ISAAC W. THORNE

SUB-COMMITTEES—SAN FRANCISCO

Clinical Program Committee (General Surgery)—ALSON R. KILGORE, Chairman; EDMUND BUTLER, THOMAS F. MULLEN.

Clinical Program Committee (Eye, Ear, Nose and Throat Surgery)—JOSEPH L. MCCOOL, Chairman; H. B. GRAHAM, WARREN D. HORNER.

Transportation Committee—EMILE F. HOLMAN, Chairman; G. D. DELPRAT, MILLARD R. OTTINGER.

Public Meeting Committee—JACOB C. GEIGER, Chairman; HAROLD M. BEHNEMAN, EDGAR L. GILCREEST, JACQUES P. GRAY, LEON WILBOR.

Golf Committee—LEROY BROOKS, Chairman; HARRY ALDERSON, JAMES MORGAN, FRANK SHEEHY.

HOSPITAL REPRESENTATIVES

SAN FRANCISCO—GENERAL SURGERY

Franklin Hospital—ERNST GEHRELS, LEROY BROOKS.

French Hospital—ASA COLLINS, EDMUND MORRISSEY.

Hospital for Children—LEROY ABBOTT, ALMA PENNINGTON.

Letterman General Hospital—R. F. METCALFE.

Mary's Help Hospital—ISAAC THORNE, RAYMOND MILLZNER, EVERETT CARLSON, DUDLEY SMITH.

Mount Zion Hospital—HAROLD BRUNN, FRANKLIN HARRIS, ALBERT L. BROWN.

St. Francis Hospital—JAMES O'CONNOR, CALVIN A. WALKER.

St. Joseph's Hospital—ALSON R. KILGORE, J. MINTON MEHERIN.

St. Luke's Hospital—G. D. DELPRAT, OTTO PFLUEGER.

St. Mary's Hospital—THOMAS E. BAILLY, PHILIP ARNOT, DANIEL SOOY.

San Francisco Hospital—Stanford University Service: LEO ELOESSER, H. MATTHEWSON, L. ROGERS. University of California Service: HAROLD BRUNN, C. LATIMER CALLANDER, GEORGE K. RHODES.

Shriners' Hospital for Crippled Children—SYLVAN HAAS.

Southern Pacific General Hospital—WILLIAM WASHBURN, FRANK R. GIRARD.

Stanford University Hospitals—EMILE HOLMAN, PHILIP K. GILMAN, FREDERICK REICHERT.

Stanford University School of Medicine—LOREN CHANDLER, EMMET RIXFORD.

United States Marine Hospital—MARK J. WHITE, RICHEY L. WAUGH.

University of California Hospitals—HOWARD C. NAFFZIGER, H. GLENN BELL.

University of California Medical School—LANGLEY PORTER, WALLACE TERRY.

Veterans Administration Hospital—P. E. JOHNSON, BERTHEL H. HENNING, JOHN A. KENNEDY.

SAN FRANCISCO—SURGERY OF THE EYE, EAR, NOSE AND THROAT

French Hospital—EDWARD C. FABRE-RAJOTTE, VICTOR D'ERCOLE.

Hospital for Children—GEORGE HOSFORD.

Letterman General Hospital—A. E. SCHLANSER, H. C. MAXWELL.

Mary's Help Hospital—FRANK HAND, J. W. CRAWFORD.

Mount Zion Hospital—FRANK RODIN, HERBERT COHN.

St. Francis Hospital—CONSTANTINE BRICCA, AUBREY RAWLINS.

St. Joseph's Hospital—ROY PARKINSON.

St. Luke's Hospital—AMBROSE E. EDGERTON, CHARLES BATES.

St. Mary's Hospital—FRANCIS CONLAN, STANLEY BURNS.

Southern Pacific General Hospital—WILBUR SWETT.

Stanford University Hospitals—EDWARD SEWELL, HANS BARKAN, HARRINGTON GRAHAM, LOUIS MORRISON.

United States Marine Hospital—RAE ASHLEY.

University of California Hospitals—WALLACE SMITH, FRED C. CORDES, ROBERT C. MARTIN, C. ALLEN DICKEY.

Veterans Administration Hospital—J. J. CAVINESS, OTTO BARKAN.

OAKLAND EXECUTIVE COMMITTEE

OLIVER D. HAMLIN, Chairman
CHARLES A. DUKES, Vice Chairman
WHITFIELD CRANE, Secretary

Clinical Program Committee—LEMUEL P. ADAMS, WARREN B. ALLEN, HARRY H. APPLEDORN, FRANK S. BAXTER, JULIAN J. BENTON, WHITFIELD CRANE, CHARLES A. DUKES, SUMNER EVERINGHAM, CHANNING HALL, OLIVER D. HAMLIN, HENRY W. HARDING, HAROLD H. HITCHCOCK, T. O. LAKE, THEODORE C. LAWSON, ERGO MAJORS, W. EARL MITCHELL, GEORGE MCCLURE, ALBERT MEADS, RAYMOND J. NUTTING, CLAIR RASOR, DEXTOR N. RICHARDS, MILTON H. SHUTES, H. R. SMITHIES, CLIFFORD SWEET, FRANK WALSH, DON D. WEAVER.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY, GYNECOLOGY, OBSTETRICS, ORTHOPEDICS, UROLOGY, SURGICAL PATHOLOGY, ETC.

OPERATIVE CLINICS IN SAN FRANCISCO HOSPITALS—DAILY

SAN FRANCISCO HOSPITAL

University of California Service

HAROLD BRUNN, GEORGE K. RHODES, A. R. KILGORE, C. L. CALLANDER, S. H. MENTZER, A. L. BROWN, H. W. STEPHENS, F. S. FOOTE, M. W. DEBENHAM, H. M. BLACKFIELD and L. GOLDMAN. General surgery.

F. HINMAN, C. JOHNSON, S. OLSEN, L. PLAYER, J. J. SULLIVAN, W. A. CARROLL and T. O. POWELL. Urological operations.

W. G. MOORE, A. M. VOLLMER and M. SCHULZE. Gynecological operations.

H. W. FLEMING, H. A. BROWN and L. B. LAWRENCE. Neurosurgical operations.

LEROY ABBOTT, F. G. LINDE, F. C. BOST, W. J. COX and K. O. HALDEMAN. Orthopedic operations.

Stanford University Service

LEO ELOESSER. Lobectomy for lung tumor; flap operation for tuberculous empyema; disarticulation at knee joint.

W. L. ROGERS. Apicolysis (paraffin fill).

J. M. MEHERIN. Gastric resection (Billroth I).

EDMUND BUTLER. Pyloroplasty for peptic ulcer; atresia of colon, congenital.

J. CLINE. Exploration of biliary duct.

C. MATHEWSON. Open reduction of spiral fracture of the tibia; sequestrectomy for tuberculosis of the pelvis.

D. KING. Nonunion of carpal scaphoid.

M. R. OTTINGER. Resection for carcinoma of the colon.

E. TOWNE. Laminectomy for decompression of cauda-equina.

E. MORRISSEY. Removal of cord tumor.

L. REYNOLDS. Cystectomy for carcinoma of the bladder.

G. HARTMAN. Suprapubic prostatectomy.

L. MICHAELSON. Plastic on kidney pelvis.

R. CRAIG. Nephrectomy.

K. SCHAUPP. Removal of fibromyoma of the uterus.

A. PETTIT. Vesicovaginal fistula.

H. VON GELDERN. Perineal repair operation.

C. COOLEY. Operation for pelvic inflammatory disease.

R. DUNN. Hyam's conization of the cervix.

D. DALLAS. Vaginal hysterectomy.

UNIVERSITY OF CALIFORNIA HOSPITAL

HOWARD C. NAFFZIGER, D. JONES, JR., H. BROWN and R. AIRD. Neurosurgical operations.

HAROLD BRUNN and H. STEPHENS. Thoracic surgery.

R. ABBOTT, F. BOST, K. HALDEMAN and W. KEYS. Orthopedic surgery.

A. MAXWELL, M. SCHULTZE, D. MORTON and C. HAYDEN. Gynecological and obstetrical operations.

FRANK HINMAN, C. JOHNSON, S. OLSEN and B. WAYMAN. Urological operations.

W. S. TERRY. General surgical operations; thyroidectomy.

H. SEARLS and H. GLENN BELL. Carcinoma of colon; cholecystectomy; popliteal aneurism.

C. ROSSEN. Appendectomy; hernioplasty.

M. S. WOOLF. Carcinoma of rectum and lower bowel.

F. FOOTE. Partial obstruction, new operations.

E. O. BARTLETT. Carcinoma of breast.

STANFORD UNIVERSITY HOSPITAL

EMMET RIXFORD, EDMUND BUTLER, R. GILMAN, L. CHANDLER, EMILE HOLMAN and S. BUNNELL. General abdominal surgery; gastro-intestinal surgery; herniæ.

EMILE HOLMAN and LEO ELOESSER. Thoracic surgery.

F. REICHERT and E. TOWNE. Neurosurgical operations.

R. GILMAN, EMMET RIXFORD, EMILE HOLMAN. Thyroid, biliary tract, liver and pancreas surgery.

A. L. FISHER, D. KING and M. MENSOR. Orthopedic operations.

J. DILLON and L. REYNOLDS. Urological operations.

L. EMGE, H. A. STEPHENSON, C. FLEUHMANN, P. E. HOFFMAN, G. CRAIG and W. STEVENS. Gynecological and obstetrical operations.

F. REICHERT and EMILE HOLMAN. Cardiac conditions, circulatory diseases.

C. B. PALMER and R. BURROWS. Injections, anæsthesia.

R. A. SCARBOROUGH. Proctological operations.

A. DAVIS and S. BUNNELL. Plastic surgery; industrial cases; skin diseases.

MARY'S HELP HOSPITAL

R. MILLZNER. Radical neck dissection.

E. CARLSON and C. C. MCCRAE. Abdominal operations.

M. MENSOR and L. PARKER. Orthopedic surgery.

M. VECKI. Urological operations.

H. VON GELDERN and A. SCHMIDT. Gynecological and obstetrical operations.

U. S. MARINE HOSPITAL

ROBERT A. JONES. Excision of pilonidal sinus and rectal operation; cholecystectomy and autoplasty (Gillies tubular flap); Dupuytren's contracture.

RICHEY L. WAUGH. Inguinal hernioplasty using pedicled fascial strips; operation for sciatica (Heyman); arthrotomy of the knee with excision of internal semilunar cartilage; bunion operation (Peabody); phrenic neurectomy.

FLETCHER C. STEWART. Transurethral resection of prostate.

ST. FRANCIS HOSPITAL

G. B. O'CONNOR. Plastic surgery: Reconstruction of face after burns; rib cartilage transplant to the nose; removal of nasal hump; cleft palate; reconstruction surgery of the hand.

W. W. WASHBURN. Thyroidectomy.

L. R. REYNOLDS and O. NOLAN. Prostatectomy, ureteral transplants.

ST. LUKE'S HOSPITAL

ALANSON WEEKS, G. D. DELPRAT, PAUL CASTELHUN, A. H. ROSBURG, OTTO H. PFLUEGER, DR. SULLIVAN, DR. MOORE and ALBERT M. VOLLMER. General surgical operations.

GEORGE J. MCCHESNEY, RUDOLPH L. DRESEL and DR. COX. Orthopedic operations.

L. P. PLAYER, HERBERT D. CRALL and MILEY B. WESSON. Urological operations.

J M. MORGAN. Proctological operations.

MOUNT ZION HOSPITAL

HAROLD BRUNN. Total thyroidectomy for cardiac disease.
F. I. HARRIS. Torek operation for undescended testicle, first and second stage procedures; resection for lesions of descending colon; radical mastectomy, Percy cautery and endotherm.
A. L. BROWN. Plastic repair of pendulous breasts; herniotomy.
W. WALDEYER. Appendectomy.
M. GROPER and A. WHITE. Blood transfusion, citrate, Linderman and Unger methods.
L. HOFFMAN. Hysterectomy.
L. D. PRINCE, A. SIRBU and DAVID CHARMAK. Radical clavectomy; fracture of os calcis.
H. BLACKFIELD. Plastic correction of congenitally protruding ears.
HAROLD BRUNN and A. L. BROWN. Phrenic avulsion; thoracoplasty.
A. ZOBEL and D. A. SUSNOW. Electrocoagulation of tumors of the rectum; hæmorrhoidectomy under local infiltration.
L. C. JACOBS. Calculi of the urinary bladder; transurethral prostatectomy.
H. A. R. KREUTZMANN. The problem of urinary lithiasis; nephrotomy; pyelotomy; nephrectomy.
B. STRAUSS and M. POLSE. Hydrocele operation; plastic operation for phimosis; cystoscopy.
A. EPSTEEN. Injection of vas deferens for chronic epididymitis.
R. K. SMITH. Classical cæsarean section.
A. BERNSTEIN. Demonstration of cervical repair immediately following delivery.
F. PEARL. Muscle splitting extraperitoneal lumbar sympathetic ganglionectomy, a new approach; muscle splitting posterior cervicodorsal sympathetic ganglionectomy.
EDWARD H. BOLZE, HERBERT H. SCHULTZ and DR. LAZAR. Demonstration of introduction of anæsthesia by avertin, evipal, gas and oxygen, spinal.

FRANKLIN HOSPITAL

E. GEHRELS. Gastro-intestinal surgery.
L. BROOKS. Abdominal operations.
J. SALE, V. DILLON, W. MONTGOMERY and W. COX. Industrial surgery and orthopedics.
G. W. PIERCE and G. O'CONNOR. Reconstruction surgery of hand, face and neck after burns; repair of eyelids; correction of flexion contractures.

ST. JOSEPH'S HOSPITAL

ALSON KILGORE, J. M. MEHERIN, F. SHEEHY and C. E. SMITH. General surgical operations.
R. SOTO-HALL and K. HALDEMAN. Orthopedic operations.
E. MORRISSEY. Neurosurgical operations.
H. VON GELDERN. Gynecological operations.
T. GIBSON. Urological operations.

FRENCH HOSPITAL

F. A. LOWE. Fractured humerus; internal derangements of knee joint.
G. W. PIERCE and G. O'CONNOR. Removal of nasal hump.

ST. MARY'S HOSPITAL

T. E. BAILLY. Gastric surgery; gastrectomy.
RODNEY YOELL. Gall-bladder surgery; cholecystectomy.
D. SOOY. Surgical intervention for duodenal ulcer.
C. P. MATHE. Nephropexy for nephroptosis.
E. TOPHAM. Inguinal hernia.
JUSTIN MCCARTHY. Industrial emergency cases.
EDMUND BUTLER. Surgery of the colon.
GEORGE K. RHODES. Emergency surgery.
EDMUND MORRISSEY. Sympathectomy for Raynaud's disease.
PHILIP ARNOT. Obstetrical surgery.
T. GIBSON. Nephrectomy.
W. FAULKNER. Bronchoscopic diagnosis of lung abscess.
J. LOUTZENHEISER. Orr treatment for delayed bony union.

LETTERMAN GENERAL HOSPITAL

R. F. METCALFE. Cauterization of cervix; perineorrhaphy; suspension of uterus; gastrojejunostomy; cholecystectomy; hæmorrhoidectomy; colostomy for rectal carcinoma; cæsarean section.
F. L. COLE. Hernia; appendectomy; inguinal hernia; ventral hernia.
H. S. BLESSE. Genito-urinary operations; electrical resection.
B. S. BURNET. Thoracotomy for empyema; orthopedic operations; bone graft; open reduction of tibia; excision of cartilage, knee.
P. E. DUGGINS. Curettage and insertion of Carsten pessary for sterility.

SOUTHERN PACIFIC HOSPITAL

W. B. COFFEY and J. D. HUMBER. Superior cervical sympathectomy for angina pectoris, moving picture demonstration in natural color.
C. MATHE and T. GIBSON. Transurethral prostatectomy
E. GREENWOOD. Cholecystectomy.
F. R. GIRARD. Inguinal hernia; ambulant treatment by injection.
C. WALKER and J. BOHM. Open reduction of fractures.
OSCAR F. NOLAN and THOMAS E. GIBSON. Suprapubic prostatectomy.
W. W. WASHBURN. Thyroid surgery.

SHRINERS' HOSPITAL

SYLVAN L. HAAS. Longitudinal osteotomy; transplantation of muscles in paralysis; stabilization of foot; fusion of spine; lengthening of leg; congenital dislocation of hip; Sever operation for obstetrical paralysis with transplantation of teres major; fusion of hip; plastic operation.

HOSPITAL FOR CHILDREN

C. HOAG. Thyroidectomy.
MURIEL E. EDWARDS. Thyroglossal duct cyst.
ALMA PENNINGTON. Supravaginal hysterectomy; total hysterectomy; vaginal plastic.

VETERANS ADMINISTRATION

Staff. Coffey operation for carcinoma of the rectosigmoid, second state; gastrectomy.

CLINICAL DEMONSTRATIONS IN SAN FRANCISCO HOSPITALS—DAILY

GENERAL SURGERY

A. S. WHITE and F. I. HARRIS. Injection treatment of hernia.

HAROLD BRUNN. Appendicitis.

S. R. SHERMAN. Rupture of the spleen.

J. HOMER WOOLSEY and H. GLENN BELL. Splenectomy.

WALTER B. COFFEY. Inspection of an industrial medical and surgical center; ward rounds; demonstration of cases; postoperative treatment.

EMMET RIXFORD. Knotty problems in industrial surgery: Traumatic carcinoma of breast; ruptured heart; traumatic thrombosis of iliac and other large veins.

EMILE HOLMAN. Operative cure of recurrent and direct inguinal hernia.

GEORGE K. RHODES. Hematogenous perinephric abscess; peritonitis and drainage.

M. W. DEBENHAM. Aseptic meningitis following spinal anæsthesia.

A. L. BROWN. Pulmonary embolectomy, motion picture demonstration of the Trendelenburg operation on cadaver.

H. BRODIE STEPHENS. Subphrenic abscess; vaccination of the pleural and abdominal cavities.

WALTER BIRNBAUM. Tendon repair; acute gonococcic tenosynovitis.

ALSON R. KILGORE, OTTO H. PFLUEGER and R. S. STONE. Treatment of breast cancer, end results.

OTTO H. PFLUEGER. Soft tissue sarcomas.

ALSON R. KILGORE. Cystic disease of the breast; cancer.

C. L. CALLANDER. Gas bacillus infection; new amputation of thigh in lower third; treatment of septic joints.

EDMUND BUTLER. Emergency surgery.

EDMUND BUTLER, L. R. REYNOLDS, L. H. GARLAND and J. B. MCNAUGHT. Old healed ruptured bladders, diagnostic difficulties and value of X-ray in diagnosis; X-ray in differential diagnosis of acute abdomen.

EUGENE S. KILGORE. Circulatory disease in differential diagnosis of acute abdomen.

CARLETON MATHEWSON, JR. and J. B. MCNAUGHT. Lymphogranuloma inguinale.

A. S. MUSANTE. Postoperative infections.

I. W. THORNE. Squamous and basal cell carcinoma of face and neck, pathology, diagnosis and treatment.

Z. E. BOLIN. Biopsies and tumor surgery; mixed tumors of the parotid.

EVERETT CARLSON. Carotid body tumors; splenectomy, indications and technique.

FRANK E. STILES. Treatment of varicose veins.

J. F. RICKARD. Intestinal obstruction.

SURGERY OF THE THYROID

WILLIAM J. KERR, HENRY H. SEARLS, JANE T. PAXSON and R. S. STONE. Activities of the thyroid committee of the University of California Hospital with follow-up studies after various lines of treatment.

HENRY H. SEARLS, E. I. BARTLETT and C. L. CONNOR. Chronic diffuse thyroiditis.

HENRY H. SEARLS and JANE T. PAXSON. Clinical picture of toxic adenoma with normal or lowered metabolic rate.

WILLIAM J. KERR. The heart in parathyroidism.

M. L. MONTGOMERY. Lingual thyroid.

THEODORE ALTHAUSEN. Surgical implications of hepatic damage in thyrotoxicosis.

R. J. MILLZNER. Parathyroid damage during thyroidectomy.

GENITO-URINARY SURGERY

FRANK HINMAN, CLARK M. JOHNSON and BRENT WEYMAN. Tumors of the testicle, pathology, demonstration of hormone tests, end-results; uretero-intestinal anastomosis, experimental work, drawings and motion picture demonstration, demonstration of patients; prostatism, pathology, indications for different types of surgery, end-results by different methods.

C. P. MATHE. Surgery of the prostate.

T. E. GIBSON. Newer aspects of renal tuberculosis.

L. P. PLAYER and H. D. CRALL. Gracilis transplantation for urinary incontinence.

MILEY B. WESSON. Conservative surgical treatment of nephrolithiasis.

L. C. JACOBS. Calculi of urinary bladder; suprapubic and transurethral prostatectomy.

H. A. R. KREUTZMANN. Urinary lithiasis, nephrotomy, pyelotomy and nephrectomy.

BERNARD STRAUSS and M. L. POLSE. Operation for hydrocele; plastic operation for phimosis.

A. EPSTEEN. Injection of vas deferens for chronic epididymitis.

J. V. LEONARD and GEORGE W. HARTMAN. Demonstration in urology.

C. P. MATHE and T. E. GIBSON. Transurethral prostatectomy.

T. E. GIBSON and O. F. NOLAN. Suprapubic prostatectomy.

J. R. DILLON. Treatment of chronic pyelitis and pyelonephritis; treatment of cancer of prostate; technical improvements in surgical treatment of undescended testicle.

W. E. STEVENS. Unusual pathological conditions of the urinary tract in women.

EDGAR POTH. A new aseptic technique for uretero-enterostomy; mechanism of ascending infection of the urinary tract; experimental observations.

SIDNEY OLSEN. Tuberculosis of the genito-urinary tract; urinary calculi.

CLARK M. JOHNSON. Trauma of the genito-urinary tract; infections of the genito-urinary tract; renal and para-renal infections; renal anomalies.

L. P. PLAYER. Kidney lavage.

W. A. CARROLL. Ureteral lithiasis; rupture of kidney.

T. O. POWELL. Newer knowledge of tumors of the testicle with special reference to gonadotropic hormone excreted in the urine.

M. R. OTTINGER, LLOYD R. REYNOLDS and J. B. MCNAUGHT. Torek operation for undescended testicle; torsion of testicle.

GEORGE W. HARTMAN. Hematuria and pyuria; renal tuberculosis.

W. A. SUMNER. Relationship of chronic infections to lesions of the genito-urinary tract.

LEWIS MICHELSON. Obstruction of the neck of the bladder in the female.

R. GLENN CRAIG. Ureteral pain of obscure origin.

MORRELL VECKI. Renal movability.

SURGERY OF INFECTIONS

A. S. WHITE. Treatment of staphylococcus infections with staphloid.

S. A. GOLDMAN. Studies on staphylococcus infections.

F. J. MCCARTHY. End-results in infections of the hand.

B. F. ALDEN. Relation of focal infection to Wassermann-fast lues.

ORTHOPEDIC SURGERY

GEORGE J. MCCHESNEY, W. COX and R. L. DRESEL. Fracture of neck of femur; treatment without external splinting.

L. D. PRINCE, A. B. SIRBU and D. D. CHARMACK. Fractures of os calcis; replacement of tibial shaft by fibula following osteomyelitis; treatment of bursitis.

R. L. WAUGH. Clinical demonstration of Roger Anderson "Ought-O-Matic" splint and skeletal traction and countertraction methods applicable to Thomas or Hodgen splints.

LEROY C. ABBOTT. The shoulder joint.

JOHN B. DE C. M. SAUNDERS. The shoulder joint.

J. F. RINEHART. Vitamin C deficiency in arthritis.

KEENE HALDEMAN and JOHN B. DE C. M. SAUNDERS. Demonstrations of bone growth.

FRANCES BAKER. Heat therapy.

F. A. LOWE. Internal derangements of knee joint, clinic and motion picture demonstration; fracture of humerus, clinic and motion picture demonstration.

J. J. LOUTZENHEISER. Arthrodesis of foot.

S. L. HAAS. Application of Hibbs-Risser plaster for scoliosis; results of treatment for scoliosis; results of tendon transplantation; Leggs-Perthes disease.

J. J. SALE, W. O. MONTGOMERY, V. M. DILLON and W. J. COX. Industrial surgery and orthopedics.

J. H. O'CONNOR. Reduction of complicated fractures, closed methods, demonstration of cases, indications for open reduction.

C. A. WALKER. End-results of open reduction of fractures; treatment of compression fractures of spine, 250 cases; fractures of clavicle and patella.

W. W. WASHBURN. Disabilities following fractures, factors influencing period of recovery.

LEONARD W. ELY. Arthritis of the hip.

D. KING. Functional anatomy and pathology of the shoulder joint.

A. L. FISHER. Treatment of flat feet.

MERRILL C. MENSOR. Osteogenic sarcoma of spine; relation of bacteriophage to the Orr treatment of osteomyelitis.

NELSON J. HOWARD. Traumatic lesions of bursæ, tendons and muscles.

LEON PARKER. Familial tendencies in Paget's disease.

D. KING. Treatment of chronic sclerosing osteomyelitis.

F. G. LINDE. Compression fractures of spine; non-union of fractures.

F. C. BOST. Hibbs-Risser treatment of scoliosis; dislocation of carpal semilunar; fracture of ankle; ligamentous tears of ankle; treatment of fracture of os calcis.

W. J. COX. Internal derangement of knee joint, rupture of ligaments; treatment of fracture of femoral neck with Smith-Peterson nails.

KEENE O. HALDEMAN. Pathology of acute osteomyelitis; pathology of chronic infections of bone.

RALPH SOTO-HALL and KEENE O. HALDEMAN. Fracture dislocation of cervical spine; Duncan's traction apparatus.

PAUL E. JOHNSON, B. H. HENNING and JOHN A. KENNEDY. Disability ratings of Veterans Administration for orthopedic conditions of the extremities.

CARLETON MATHEWSON, JR. and J. B. MCNAUGHT. Treatment of spiral fractures of tibia; open and closed methods of treatment of fractures of extremities; tuberculosis of pelvis.

D. KING, J. M. MEHERIN and R. A. SCARBOROUGH. Fracture of carpal scaphoid; surgical approaches to bones and joints; Orr method of treatment of osteomyelitis.

NELSON J. HOWARD. Fractures of the upper end of the humerus, motion picture demonstration.

MERRILL C. MENSOR and LEON PARKER. Unusual fractures of the spine; treatment of osteomyelitis with surgical maggots.

C. C. MCRAE. Injuries of small bones of the hand.

EDGAR L. GILCREEST. Problems in treatment of fractures.

THORACIC SURGERY

HAROLD BRUNN, A. L. BROWN, H. ROSENBLUM and J. J. SAMPSON. Symposium on surgery of the heart with particular reference to adhesive pericarditis.

LEO ELOESSER, PHILIP H. PIERSON, W. L. ROGERS, W. G. BURKHARD, DAVID A. WOOD, W. R. CLARK and L. H. GARLAND. Various types of bronchial stenosis; mycotic infections of the lung; tumors of the lung; empyema.

EMILE HOLMAN. Technical improvements in partial selective thoracoplasty; resection of transverse process; resection of scapula; ligation of the pulmonary artery as a therapeutic measure in pulmonary hæmorrhage; carcinoma of lung simulating inflammatory disease.

HAROLD BRUNN, SIDNEY J. SHIPMAN, H. BRODIE STEPHENS, A. L. BROWN, M. W. DEBENHAM and A. GOLDMAN. Lung suppurations; empyema; artificial pneumothorax; phrenic avulsion; thoracoplasty.

ALANSON WEEKS and G. D. DELPRAT. Thoracoplasty.

RAY KISTLER. Diaphragmatic hernia.

SIDNEY J. SHIPMAN. Pneumothorax in pneumonia.

W. B. FAULKNER, JR. Bronchiectasis; treatment of chest injuries.

A. L. BROWN. Collapse therapy in pulmonary tuberculosis.

S. SHERMAN. Lymphoblastoma of mediastinum.

C. A. WALKER. Phrenicectomy for pleuropericardial adhesions.

A. GOLDMAN. Staphylococcic infections of the lung; chemotherapy in tuberculosis.

T. F. MULLEN. Antethoracic œsophagoplasty.

MARY E. MATHES. Experimental study of the effect of various pathological conditions upon the dual blood supply of the lungs.

EDGAR POTH. A simple apparatus for tidal and siphon irrigation and its application in treatment of empyema.

DAVID A. WOOD and MARY E. MATHES. Exhibit of clinical and experimental observations on the dual blood supply of the lungs in various pathological states.

NEUROSURGERY

HOWARD C. NAFFZIGER. Late results in the treatment of malignant exophthalmos; brain tumors; factors influencing recovery after peripheral nerve injury; cervical ribs and "the scalenus syndrome without cervical ribs."

HOWARD W. FLEMING. Subdural hematomata; cerebrospinal rhinorrhea; relief of intractable pain; cranial approach for orbital tumors; craniocerebral injuries.

EDMUND MORRISSEY. Neurologic clinic on lesions of the cauda equina; diagnosis and treatment of subdural hæmorrhage; diagnosis of subdural hæmorrhage.

O. W. JONES, JR. Spinal cord tumors.

H. A. BROWN. Low back injuries; spinal cord injuries.

E. B. TOWNE. Treatment of acute head injuries.

F. L. REICHERT. Neuralgias of cranial nerves, demonstration of patient and lantern slides.

ROBERT AIRD. Encephalography, clinical and experimental; intradural alcohol injections for intractable pain.

E. B. TOWNE, E. MORRISSEY, J. W. WOLFSOHN and D. WOOD. Surgical lesions of the spinal cord; dynamics of epilepsy.

L. B. LAWRENCE. Spinal cord tumors; tumors of cauda equina.

LEE HAND. Regeneration of peripheral nerves of hand.

GYNECOLOGY AND OBSTETRICS

WILLIAM G. MOORE. Endometriosis; fibromyomata of uterus.
A. M. VOLLMER. Rubin's insufflation test; trichomonas vaginalis.
R. K. SMITH. Classical cæsarean section, motion picture demonstration.
FRANK LYNCH, ALICE MAXWELL and R. S. STONE. Uterine cancer, follow-up, X-ray therapy, radium therapy.
MARGARET SCHULZE. Special ovarian tumors.
A. H. HEALD and ALICE MAXWELL. X-ray pelvimetry, direct method.
PHILIP H. ARNOT. Conduct of labor in posterior position.
LUDWIG EMGE. Dysmenorrhea, causes and treatment; sterility, diagnosis and treatment.
C. F. FLUHMANN, P. E. HOFFMAN and GERTRUDE F. JONES. Endocrinological aspects of gynecology, modern methods of diagnosis, blood and urine hormone tests, biopsy of endometrium, hormone therapy.
A. V. PETTIT. Results of hyperpyrexia in treatment of acute and chronic pelvic inflammatory disease.
LUDWIG EMGE. Radiation therapy of carcinoma of cervix, methods and end-results.
A. M. VOLLMER. Treatment of abortions.
MARGARET SCHULZE. Multiple pregnancies; pyelitis with pregnancy; hydatidiform mole and chorio-epithelioma; cardiac disease with pregnancy.
KARL L. SCHAUPP. Fibromyoma of the uterus.
HANS VON GELDERN. Plastic operations on pelvis.
C. L. COOLEY. Demonstration of gynecological cases.
R. D. DUNN. Treatment of incomplete abortions.
D. A. DALLAS. Operations in obstetrics.
BEVERLY SIMPSON. Separated placenta.
ADOLPH E. SCHMIDT. Uterine bleeding.

SURGERY OF THE GASTRO-INTESTINAL TRACT

HAROLD BRUNN. Cancer of the rectum.
F. I. HARRIS. Cicatrizing (chronic) enteritis (regional ileitis); treatment of appendix stump, noninversion.
FRED H. KRUSE. The more common complications of peptic ulcer.
E. J. BEST, F. H. KRUSE, THEODORE ALTHAUSEN and RALPH RABINOWITZ. Postoperative care of intestinal conditions.
M. F. CUNHA. Primary duodenitis; end-results of ulcer cases, types of operation, causes of recurrence.
LEON GOLDMAN and THEODORE ALTHAUSEN. Pseudo perforation of peptic ulcer.
J. HOMER WOOLSEY and H. GLENN BELL. Carcinoma of stomach.
M. L. MONTGOMERY and JOSEPH M. SWINDT. Acute intestinal obstruction, experimental and clinical.
H. GLENN BELL. Subacute intestinal obstruction, localized type (chronic cicatrizing enteritis).
H. GLENN BELL and LEON GOLDMAN. Congenital lesions, tumors, diverticula of small bowel.
M. S. WOOLF, LEON GOLDMAN and H. GLENN BELL. Carcinoma of large bowel.
DUDLEY SMITH and J. W. MORGAN. Carcinoma of rectum.
ASA W. COLLINS. Pyloroctomy and gastro-enterostomy.
LEROY BROOKS. Diagnosis and treatment of intestinal obstruction.
ERNST GEHRELS. Radical surgery for gastric and duodenal ulcers; diverticulitis of colon; closure of colostomy; preservation of anal sphincter.
P. K. BROWN. Peptic ulcer, indications for surgical treatment.
W. W. WASHBURN. Acute perforation of peptic ulcer, complications and end-results in 100 cases.
J. A. GUILFOIL. Chronic appendicitis, end-results of operation.
J. E. BOHM. Mortality rate of operations for appendicitis.
R. A. SCARBOROUGH. Developments in surgical treatment of carcinoma of rectum, 200 cases.
EMILE HOLMAN. Causes for failure to control symptoms and to prevent gastrojejunal ulcer in gastric surgery.
GUNTHER W. NAGEL, F. L. REICHERT and MARY E. MATHES. Chronic regional enteritis, clinical, experimental.
DAVID A. WOOD. Multiple primary carcinomata of colon complicating multiple polyposis of colon.
NELSON J. HOWARD. Amebic granuloma of large bowel.
HAROLD BRUNN. Carcinoma of large bowel; carcinoma of rectum; bowel obstruction.
GEORGE K. RHODES. Spontaneous perforation of cæcum from obstruction in distal colon.
DANIEL SOOY. Choice of operation in gastric surgery.
EDWARD TOPHAM. End-results in surgery for gastric ulcer.
H. P. HILL, GEORGE BARNETT, J. M. MEHERIN, J. W. CLINE, JR., CARLETON MATHEWSON, JR., J. B. MCNAUGHT and A. C. MCKENNEY. Lesions of the upper gastro-intestinal tract; amebic infections of liver and gastro-intestinal tract.
DUDLEY SMITH. Operation for rectal fistula and hemorrhoidectomy, motion picture demonstration.

SURGERY OF THE BILIARY TRACT, LIVER AND PANCREAS

ALANSON WEEKS and G. D. DELPRAT. Common duct stone; hydatid disease of liver; granuloma inguinale.
F. I. HARRIS. Acute cholecystitis.
CARL HOAG. Reconstruction of common duct.
H. CLARE SHEPARDSON and HANS LISSER. Pancreatic dysfunction; hypoglycemia.
H. GLENN BELL and THEODORE ALTHAUSEN. Operative mortality and pre-operative management of cholecystitis, glucose therapy, Rose-Bengal and other tests.
FRED H. KRUSE and THEODORE ALTHAUSEN. Medical and surgical jaundice; cirrhosis of liver, differential diagnosis from carcinoma of stomach.
JESSE L. CARR and FREDERICK S. FOOTE. Experimental work in human jaundice.
KARL SCHMIDT. The bile salts.
EMILE HOLMAN. Postoperative and inflammatory stenosis of the bile passages.
M. W. DEBENHAM and J. M. SWINDT. Liver abscess.
STANLEY H. MENTZER. Acute cholecystitis; obstructive cholecystitis.
RODNEY A. YOELL. Gall-bladder anomalies.
T. F. MULLEN. Recurrence of symptoms after biliary tract surgery.

ENDOCRINOLOGY

R. F. ESCAMILLA. Abdominal pain of endocrine origin.
SAMUEL COHN and F. I. HARRIS. Treatment of undescended testicle by operation and glandular extracts.
LEO STANLEY. Endocrinology in a penal institution.

CIRCULATORY DISEASE

M. L. MONTGOMERY. Therapeutic venous occlusion.
C. A. NOBLE, JR. Postoperative cardiac versus circulatory collapse.

CLINICS IN ALAMEDA COUNTY HOSPITALS—WEDNESDAY

ALAMEDA COUNTY HOSPITAL

WHITFIELD CRANE and W. EARL MITCHELL—9. Carcinoma of stomach.

FRANK H. BOWLES and THEODORE LAWSON—10. Carcinoma of cæcum.

H. W. HARDING and DON D. WEAVER—11. Carcinoma of colon.

LEMUEL P. ADAMS—12. Carcinoma of breast.

SUMNER EVERINGHAM—9. Extrapleural thoracoplasty; intrapleural pneumolysis; clinic on phrenic interruption and thoracoplasty. Discussion by CHESLEY BUSH.

WARREN B. ALLEN—12. Neurosurgery.

W. F. HOLCOMB—9. Arthroplasty of hip.

L. B. BARNARD—10. Arthroplasty of shoulder.

E. N. EWER—9. Total hysterectomy; subtotal hysterectomy; discussion of obstetrical service at Alameda County Hospital.

CLARENCE A. DEPUY—11. Gynecological cancer clinic. Demonstration of intraspinal alcohol injection and presentation of cases.

ALBERT M. MEADS, LLOYD KINDALL, JOHN A. DAUGHERTY, T. I. BUCKLEY and associates—9. Perineal prostatectomy, suprapubic prostatectomy, resectoscopic prostatectomy. Operations, demonstration of cases and discussion.

Dry Clinics, 2—4:30

CHARLES A. DUKES and associates. Cancer clinic.

HAROLD H. HITCHCOCK, N. A. CARY and associates. Traumatic and orthopedic clinic; demonstration of Sweetland cast dryer, Bell table, plaster models, splints, etc.

W. H. SARGENT and C. B. BOWEN. X-ray exhibit and discussion.

GERTRUDE MOORE. Pathological exhibit and conference.

BERKELEY GENERAL HOSPITAL

Dry Clinics, 9—12

FRANK D. WALSH. Cholecystitis, observations and comments on surgical treatment.

CLAUDE H. CHURCH. Ectopic pregnancy recurring on same side.

WILLIAM W. CROSS. Polycystic kidney; nephrolithiasis; prostatic management.

J. F. CARLSON. Osteochondromatosis involving all epiphyses in one extremity; clavicle dressings.

W. W. REICH. Parathyroid disease, gross specimens and microprojections.

R. G. VAN NUYS. X-ray demonstration and discussion.

CHILDREN'S HOSPITAL

ROY NELSON—9. Demonstration of methods of treatment of œsophageal stricture due to lye.

W. W. CROSS—9. Postmortem findings in the kidneys of children, lantern slide demonstration.

CLIFFORD SWEET—9. Clinic on undescended testes: Demonstration of postoperative results; discussion of the effect of antuitrin S; demonstration of operation.

ALTA BATES HOSPITAL

Staff—9. Operations and dry clinics.

SAMUEL MERRITT HOSPITAL

WARREN B. ALLEN—9. Reconstruction of skull defects, operation and demonstration of cases.

W. F. HOLCOMB and D. D. TOFFLEMEIR—9. Orthopedic operations and demonstrations.

MARK L. EMERSON—9. Rectal surgery and presentation of cases.

FRANK H. BOWLES—9. Thyroidectomy.

W. H. SARGENT—9. X-ray demonstration and discussion of cases.

ROBERT A. GLENN—9. Pathological exhibit; demonstration of frozen section technique and specimens.

WHITFIELD CRANE—10:30. Peptic ulcer; Judd pyloroplasty.

W. EARL MITCHELL—10:30. Pelvic tumor.

HENNING KOFORD—10:30. Cholecystectomy.

CHARLES A. DUKES—11. Apicolysis, operation; demonstration of thoracic cases. Discussion by HAROLD TRIMBLE.

Dry Clinic, 2–4:30

H. N. ROWELL, A. M. SMITH, W. H. STRIETMANN, A. A. ALEXANDER, W. S. KUDER, STEWART V. IRWIN, H. GORDON MACLEAN, FLETCHER B. TAYLOR, VERNE G. ALDERSON, and HOBART ROBERTS. Symposium on pre- and postoperative care: Management of surgical jaundice and stomach cases; diabetes in surgery; traumatic and postoperative pneumonia; cardiac and renal complications; postoperative psychosis; allergy in relation to abdominal surgery; discussion and demonstration of cases.

PERALTA HOSPITAL

J. L. LOHSE—9. Cholecystectomy.

ERGO A. MAJORS—9. Carcinoma of breast, radical removal.

F. M. LOOMIS and JOHN W. SHERRICK—9. Porro cæsarean section.

CHARLES B. FOWLER—9. Orthopedic treatment of spondylolisthesis; correction of upper extremity involvement in poliomyelitis.

H. J. TEMPLETON and J. LUNSFORD—10. Electric desiccation in cutaneous malignancies.

JOHN W. SHERRICK—10. Vaginal plastic.

T. FLOYD BELL—10. Pelvic tumor.

T. C. LAWSON—11. Hernia, fascial repair.

P. N. JACOBSEN—11. Bladder surgery.

PAUL MICHAEL—9 to 1. Pathological demonstration and exhibit.

J. D. COATES—9 to 1. X-ray demonstration and exhibit.

COWELL MEMORIAL HOSPITAL

HERBERT EVANS, ROBERT LEGGE, C. A. KOFOID and associates—9. Exhibit and discussion of latest advances in endocrinology; inspection of laboratories and hospital with special reference to the systematic medical care of university students; discussion of laboratory technique with particular reference to amebiasis.

ALAMEDA SANATORIUM

J. OHANNESON—9. Hernioplasty under local anæsthesia; appendectomy.

G. R. BURKE—9. Cholecystectomy.

CHANNING HALL—10. Resection of stomach.

PROVIDENCE HOSPITAL

O. D. HAMLIN—9. Intestinal anastomosis.
J. RADFORD FEARN—9. Vaginal hysterectomy under local anæsthesia.
A. REIS—10. Cholecystectomy, new method.
THEODORE M. WELLER—10. Cæsarean section.
N. AUSTIN CARY—11. Sacro-iliac fusion.
PHILIP J. DICK—11. Posterior gastro-enterostomy.
MICHAEL TORRANO—12. Herniotomy under local anæsthesia.
S. A. JELTE—9 to 12. X-ray demonstration and discussion.

EAST OAKLAND HOSPITAL

DON D. WEAVER—9. Ulcer of the stomach.
O. R. ETTER—9. Surgery in the diabetic patient, dry clinic.
R. G. VAN NUYS—10. Dry clinic: Visceroptosis and position of the viscera in healthy young adults; X-ray demonstration.
BROOKS STEPHENS—10:30. Hallux valgus.
CLAIR RASOR—10:30. Prolapse of uterus.
ALEXANDER H. GRIFFITH—11. Carcinoma at the recto-sigmoidal juncture, Lahey technique.

SURGERY OF THE EYE, EAR, NOSE AND THROAT

CLINICS IN SAN FRANCISCO HOSPITALS—DAILY

UNIVERSITY OF CALIFORNIA HOSPITAL

Tuesday

JOSEPH L. MCCOOL, FREDERICK C. CORDES, JOSEPH W. CRAWFORD, C. ALLEN DICKEY and DAVID O. HARRINGTON—9. Ophthalmological operations.
R. C. MARTIN and FREDERICK C. CORDES—9. Toti-Mosher operation.
WALLACE SMITH, LOUIS MORRISON and EDITH STOKER—9. Otolaryngological operations.

Dry Clinics—2

FREDERICK C. CORDES. Surgery of traumatic cataracts.
J. W. CRAWFORD. Tuberculosis of the eye.
C. ALLEN DICKEY. Surgery of the vertical muscles.
DAVID HARRINGTON. Contact glasses, practical demonstration.
R. C. MARTIN and STERLING BUNNELL. Injuries and repair of the facial nerve.

Thursday

JOSEPH L. MCCOOL, FREDERICK C. CORDES, JOSEPH W. CRAWFORD, C. ALLEN DICKEY and DAVID O. HARRINGTON—9. Ophthalmological operations.
R. C. MARTIN and FREDERICK C. CORDES—9. Toti-Mosher operation.
WALLACE SMITH, LOUIS MORRISON and EDITH STOKER—9. Otolaryngological operations.

Dry Clinics—2

JOHN SAUNDERS. Regional anatomy of the mastoid and pathways of infection of the intracranium.
HOWARD C. NAFFZIGER. Brain abscess arising from middle ear and mastoid infections.
WALLACE SMITH. Phlebitis and thrombosis following middle ear and mastoid infections.

Friday—2

FREDERICK C. CORDES. Surgery of complicated cataracts.
J. W. CRAWFORD. The eye in diabetes.
C. ALLEN DICKEY. The value of orthoptic treatment.
DAVID HARRINGTON. Tobacco amblyopia and its treatment.

ST. MARY'S HOSPITAL

Thursday—2

FRANCIS CONLAN, STANLEY BURNS and FRANK HAND. Treatment of posterior sinusitis. Blood dyscrasias in relation to the ear, nose and throat. Treatment of bilateral abductor paralysis.
FRANK HAND. Radical antrum.

SAN FRANCISCO HOSPITAL

Tuesday—2

WARREN D. HORNER. External iridectomy; history, uses, technique and advantages.
C. ALLEN DICKEY and J. W. CRAWFORD. Safety procedures in cataract operations; akinesis, intra-orbital injections, lid sutures, pre-operative medication.
WARREN D. HORNER, C. ALLEN DICKEY and J. W. CRAWFORD. The use of synthetic epinephrin bitartrate in ocular therapeutics.
AUBREY RAWLINS. Osteoma of the antrum; some curious foreign bodies in the lungs; cases of recovery from otitic meningitis; extensive osteomyelitis of the frontal bone.
HARRINGTON B. GRAHAM. Foreign bodies in the lungs; stenosis of the œsophagus; cancer of the larynx.
HARRINGTON B. GRAHAM and J. M. WOLFSOHN. Extensive abscess of the meninges.
RAE ASHLEY. Treatment of tuberculosis of the larynx.

HOSPITAL FOR CHILDREN

Thursday—2

GEORGE N. HOSFORD and AVERY M. HICKS. Technique for the determination of the hydrogen ion concentration of tears. The significance of pH of tears in ocular symptoms and treatment. Indications for and results of orthoptic training. Relation of vertical imbalance of extra-ocular muscles to gastric symptoms, posture, temperament and aptitude for school work and occupation. Congenital muscle palsy. Motion picture demonstration of O'Connor technique for heterophoria and heterotropia.

Days to be Announced

GEORGE N. HOSFORD and AVERY M. HICKS—9. Extraction of congenital cataract; muscle operations, O'Connor cinch shortening for simple exotropia, simple esotropia, vertical deviations; muscle transplants for external rectus palsy, superior rectus for paralysis of the superior oblique (Jackson's technique), Motais' operation for congenital ptosis; Toti-Mosher operation for occlusion of nasolacrymal duct (with Drs. Martin and Hosmer).

STANFORD UNIVERSITY SCHOOL OF MEDICINE

Wednesday—2

FRANK RODIN. Congenital and hereditary eye defects.
AVERY HICKS. Ocular torticolis.
DOHRMANN PISCHEL. Retinal detachment, methods and results.

MOUNT ZION HOSPITAL

Tuesday

HERBERT J. COHN, EDWARD LIPSETT and JOHN SKAFF—9. Tonsil operations, local and general anæsthesia, dissection, Sluder, snaretome.

Wednesday—2

HERBERT J. COHN, EDWARD LIPSETT and JOHN SKAFF. Symposium on mastoidectomy.
GEORGE S. LACHMAN. Treatment of corneal ulcers.
CHARLES WEISS. Recently developed concepts in immunology and bacteriology of value to the ophthalmologist.
FRANK H. RODIN. Treatment of acute iridocyclitis.
G. Y. RUSK. Pathological demonstration of various eye conditions.

Thursday

Staff—9. Nasal operations: Submucous resection, electrocoagulation of turbinates, sinus surgery.

Days to be Announced

FRANK H. RODIN and GEORGE S. LACHMAN—9. Eye operations: Cataract, strabismus, plastic on eyelids.

SOUTHERN PACIFIC HOSPITAL

Thursday—2

WILBUR F. SWEET and JOHN C. WILLIAMS. Clinics and demonstration of cases.

LETTERMAN GENERAL HOSPITAL

Tuesday

A. E. SCHLANSER and HERBERT H. PRICE—9. Tonsillectomies; adenoidectomies, general anæsthesia.
HARVEY C. MAXWELL—9:30. Eye surgery, general anæsthesia: Strabismus correction by O'Connor tendon cinch and by Jamieson recession.

Wednesday

A. E. SCHLANSER—9. Tonsillectomies, local anæsthesia; nasal operations, local anæsthesia.

Thursday

HARVEY C. MAXWELL—9. Eye operations, local anæsthesia; cataract extraction, pterygium transplant, strabismus correction.

Friday

A. E. SCHLANSER, HERBERT H. PRICE and HARVEY C. MAXWELL—9. Sinus surgery, local anæsthesia; extranasal radical frontal ethmosphenoidectomy; radical maxillary sinusotomy; bronchoscopy; œsophagoscopy.

ST. LUKE'S HOSPITAL

Tuesday and Thursday

JOSEPH L. MCCOOL, C. ALLEN DICKEY, A. E. EDGERTON and CHARLES BATES—9. Ophthalmological clinic.

VETERANS ADMINISTRATION

Staff. Bronchoscopic examinations.

CLINICS IN ALAMEDA COUNTY HOSPITALS—WEDNESDAY

PROVIDENCE HOSPITAL

A. J. HOWELL—9. Reconstruction of nose.
RODERIC O'CONNOR—9. Muscle shortening; cataract; Curran operation.
GEORGE MCCLURE and NELSON KEELER—9. Ear, nose and throat surgery.
RANDOLPH SHARPSTEEN—9. Motais' operation.
ROY NELSON—9. Œsophagoscopy and bronchoscopy.
BRUCE STEPHENS, JR.—10. Cataract operation.
FRANK BAXTER—10. Ear, nose and throat operations.
MILTON H. SHUTES—10. Ear, nose and throat operations.
ALEXANDER GALBRAITH—10. Ear, nose and throat operations.
W. A. MAGRATH—11. Eye operations.
ALVIN P. WOLD—11. Eye surgery.
F. C. KRACAW—11. Ear, nose and throat surgery.
R. J. NUTTING—12. Eye operations.
P. T. LEFTWICH—12. Ear, nose and throat surgery.
FRANCIS SHOOK—12. Ear, nose and throat surgery.
SYDNEY N. PARKINSON—12. Treatment of acute and subacute paranasal sinus infections, lantern slides and motion picture demonstration.

Dry Clinic, 2—4:30 p.m.

Staff. Discussion of operative procedures, demonstration of cases, lantern and motion picture demonstration.

ALAMEDA SANATORIUM

B. M. STEPHENS—11. Cataract.

CHILDREN'S HOSPITAL

M. E. LANDO—10. Strabismus, operative treatment.